A STUDY

OF

CERTAIN PREVENTABLE DISEASES,

THE CAUSES OF DEATH

AND

METHODS OF PREVENTION

Dedicated to the Local Health Officers of Texas in appreciation of their unselfish service to humanity.

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State Health Officer

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TEXAS STATE DEPARTMENT OF HEALTH
AUSTIN, TEXAS

INDEX

Foreword

This bulletin is published with the hope that it will prove to be of benefit as a ready reference concerning the handling of the diseases which are of major concern to the health officer and other public health workers.

So far as possible, controversial points have been avoided in the preparation of this bulletin, and it is by no means intended to replace standard text books and such information as may be gathered from the current literature. It is suggested that the book might be most profitably used in connection with U. S. Public Health Service Reprint Number 1697, titled "The Control of Communicable Diseases". This periodical may be purchased from the United States Government Printing Office in Washington, D. C., at a price of five cents. In ordering these bulletins, address the Superintendent of Documents. It is suggested that the health officer might profitably place a copy of this latter bulletin in the hands of each practitioner in his community.

Every physician in the community should be familiar with the diseases that are reportable and with the provisions of the law concerning the quarantine of these diseases. In communities where a full-time health officer is available, the practitioner can reasonably expect the following upon report of a contagious disease: (1) that the premises will be visited by the health officer or his representative and that legal quarantine or isolation will be established and that the disease will be reported to the State health authority as required by law; (2) that in those conditions where epidemiological study is often fruitful of results, a case study of the origins of the disease will be made; (3) that instructions will be given the family as to the methods of disinfection and sterilization which are most practicable, and that suitable bacteriological procedures will be instituted to determine the carrier's state before releasing him from quarantine; (4) that the health officer and his representatives will in no way interfere with the treatment of the patient or patients, since this does not fall within the field of public health; (5) that all contacts to the infection will be either immunized by the health officer or urged to visit their private physician for immunization if the disease be one in which immunization is effective; (6) that the health officer will be responsible for all legal measures that are necessary to prevent quarantine violation.

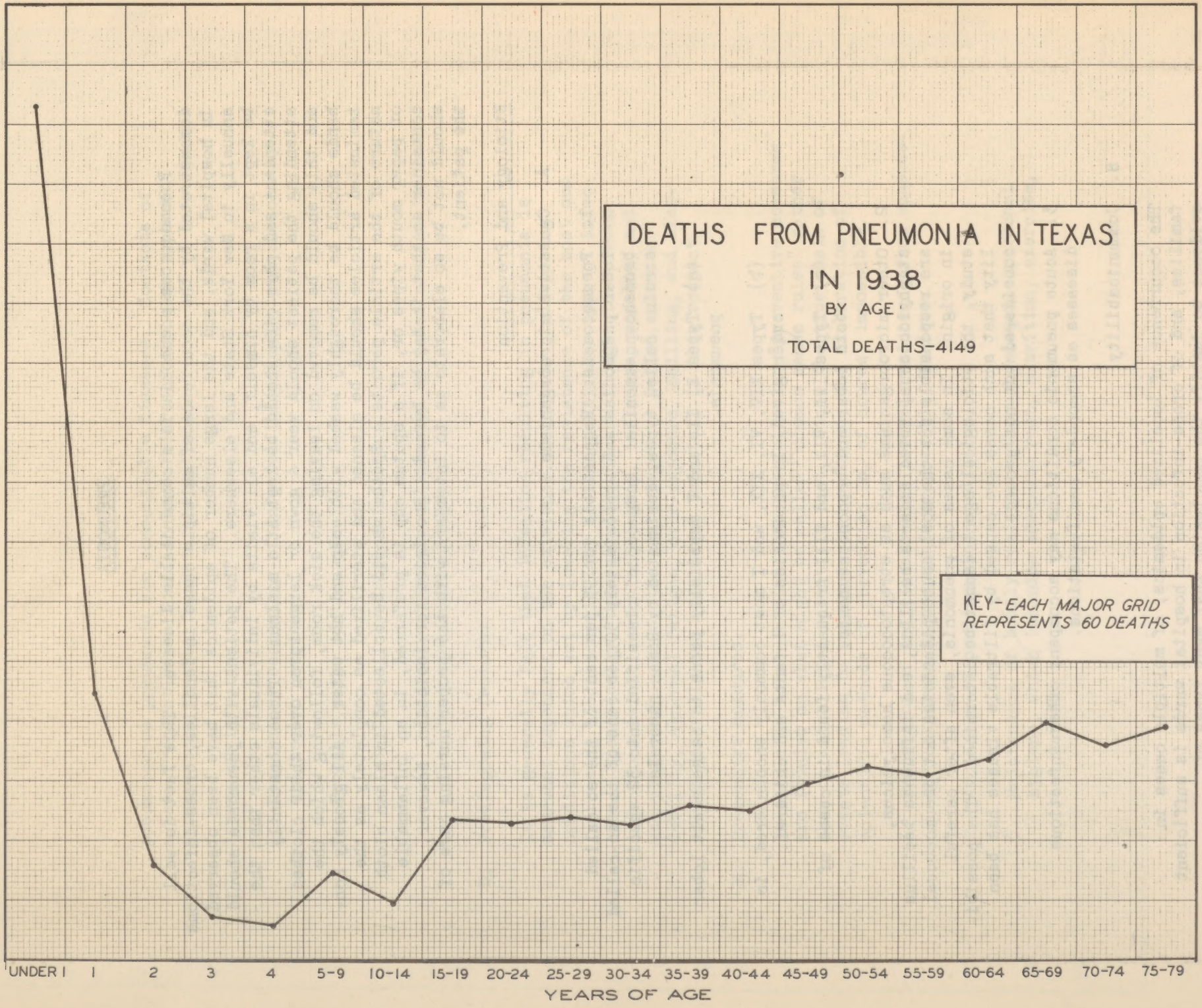
The foregoing activities relate chiefly to the duties of a full-time health officer. In communities having part-time health officers, any or all of these activities may be performed; however, many of our part-time health officers are paid little or nothing for their services and cannot afford to leave their practices to do the work normally carried on by the full-time health officer. In such communities, practitioners should report to the part-time health officer, but in many instances they will be delegated to establish quarantine and supervise disinfection, etc. It is impracticable for private practitioners to do a great deal in the way of epidemiological study, since such study usually leads to the homes of

individuals who comprise a portion of another private physician's practice. To attempt to arrive at sources of infection often results in friction between the practitioners.

While the epidemiology must usually be neglected in such cases, the bacteriological studies to determine presence or absence of the carrier's state can be, and by all means should be carried on. Since it is more to the public interest than to the individual's interest that these studies be made, they will be done in the State laboratories regardless of the economic situation of the patient. If carriers are found, these, too, should be reported to the local part-time health officer just as cases are reported.

A limited amount of epidemiological service is available from the State Health Department to communities not served by full-time health officers. In any situation which seems to warrant an epidemiological study, a request should be made for such service to the State Health Officer. However, on arrival of the State Epidemiologist in your community, it is necessary that he have some sort of records with which to begin his work. He must know who the patients are, where they live, their ages and occupations, and similar data. These data are easily collected by local practitioners on the cases in their practice, but it is extremely difficult for the visiting epidemiologist, a stranger to the community, to accumulate these data. Until they are accumulated, he cannot do any effective work.

It must be remembered that every practitioner who encounters reportable contagious disease is legally responsible for the spread of the disease to others if he does not report it to the proper local health authority. Courts have repeatedly awarded damages to individuals who have contracted a disease from an unreported case. Their cause of action is against the physician treating the case, and his only possible defense is that he had failed to make the diagnosis. Naturally, such an admission does a physician little good, even though he may escape paying damages on the basis of this plea. Once a case is reported, however, the acting physician is exonerated from all blame in the event of subsequent spread of such infection even though the local health officer takes no action to control the disease.


DEATHS FROM PNEUMONIA IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-4149
KEY- EACH MAJOR GRID REPRESENTS 60 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

PNEUMONIA

Pneumococcus pneumonia is a communicable disease. This fact is well demonstrated by the occurrence of multiple cases in families, cross-infections in hospital wards, and the large number of epidemics that have been observed annually in New York State and elsewhere. The patient with pneumonia should be kept in a room by himself, and all visits by individuals other than the attendant and physician should be kept at a minimum. Those constantly attending the patient should wear a gown or large apron over their clothes, and this should be removed on leaving the sick room, following which the hands should be thoroughly washed with soap and hot water. All grossly contaminated articles should be cleaned and sterilized as completely as the nature of the article permits. Sputum should be collected in clean cloth or paper mouth wipes or, if a sputum cup is preferred, in an inflammable container so that it may be burned. Provision for isolation, however, should not be so elaborate as to interfere with the proper nursing care of the patient.

Etiology and Prevention

A. Causative microorganisms.

1. Pneumococcus. Responsible for 95 per cent of the so-called lobar pneumonias and approximately 50 per cent of the so-called broncho-pneumonias. Pneumococci classified into 32 specific strains called types, designated by Roman numerals.

   (a) Types I, II, and V especially prone to cause adult lobar pneumonia.

   (b) Types XIV, VI, XIX, and I most commonly encountered, in order given, in pneumonias of infants and children.

   (c) Types III, VII, and VIII frequently found in cases of both lobar and bronchopneumonia.

2. Other microorganisms such as streptococcus hemolyticus, staphylococcus, Friedlander's bacillus, and influenza bacillus are responsible for the most acute pneumonias not pneumococcal in origin. In some cases of pneumonia, even after careful study, the etiological agent cannot be determined. The possibility that such cases are caused by filtrable viruses has been considered but not proved.

3. Acute pneumonic conditions may accompany other infectious diseases as secondary manifestations.

B. Communicability

The occurrence of localized epidemics, of multiple cases in families, and of cross-infection in hospital wards is sufficient evidence to justify the conclusion that many types of

pneumococcus pneumonia are communicable diseases. Contacts of a case frequently are found to be carriers of the same type of pneumococcus as that found in the patient.

C. Source of infection.

1. Respiratory secretions of infected individuals, convalescents, and health carriers.

2. Probable paths of transmission.

(a) Direct: by droplet infection.

(b) Indirect: through contaminated articles or dust in contaminated surroundings.

D. Predisposing factors.

It is generally felt that infection resulting in pneumonia is somewhat dependent on the existence of certain predisposing conditions which act by lowering the resistance of the host and increasing his susceptibility to that particular type of infection. Specifically, these conditions are thought to be such infections as the common cold, grip, influenza, measles, and whooping cough. Also, the post-operative period, chronic debility, abnormal fatigue, over-exposure, chilling, and alcoholism generally are considered important in this respect.

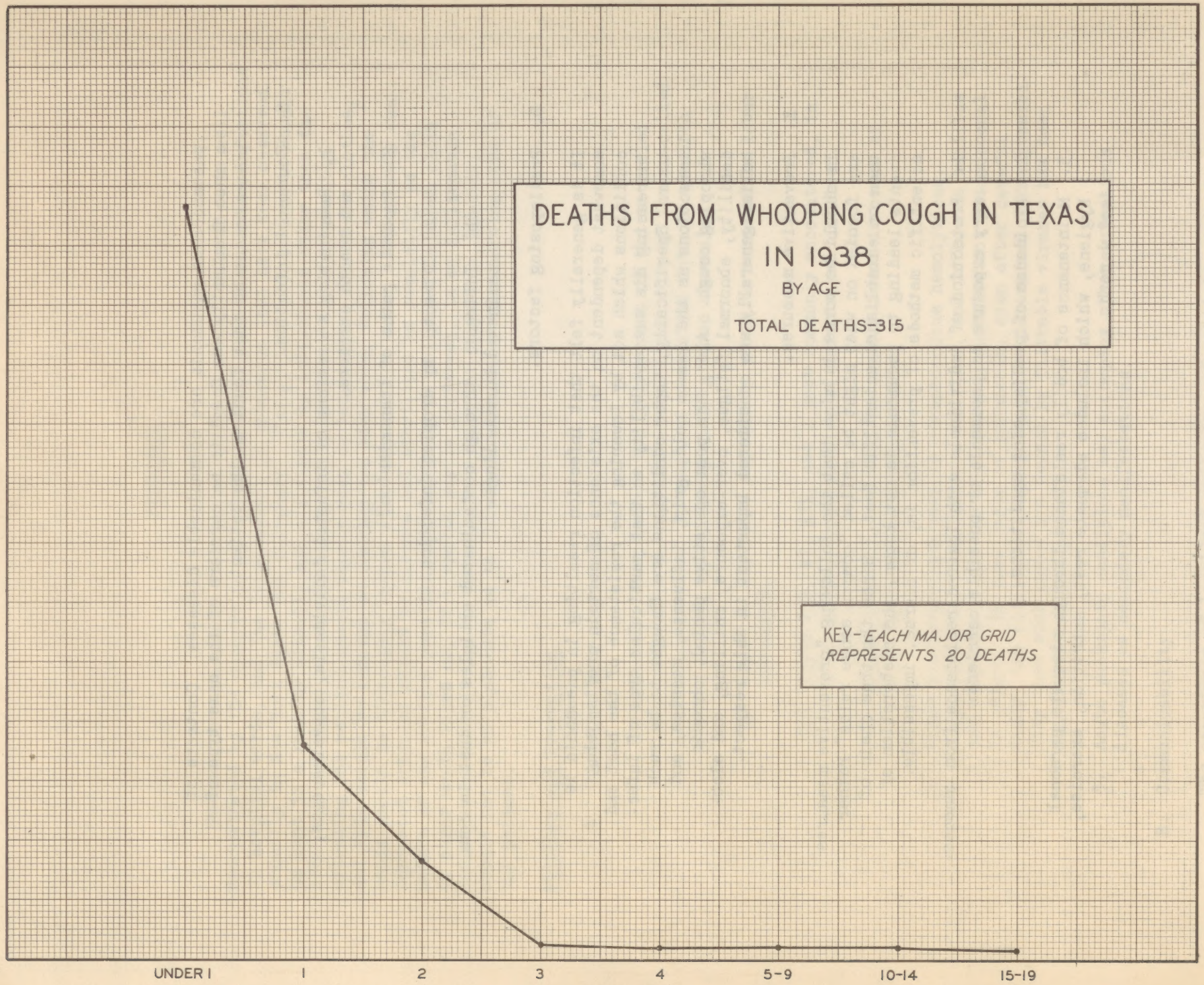
E. Preventive measures.

It should be borne in mind that the following "preventive measures" are founded on what might be called "common sense rules" rather than on established scientific fact. Since the true chain of events leading to pneumonia is not known, the elaboration of scientific methods of prevention is, of course, impossible.

1. Protection of individuals with lowered resistance from unnecessary exposure to pneumonia or possible carriers.

2. Avoidance of predisposing conditions.

3. Maintenance of bodily resistance through adequate personal hygiene, which includes adequate rest, nutrition, exercise, and warmth.


DEATHS FROM WHOOPING COUGH IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-315
KEY- EACH MAJOR GRID
REPRESENTS 20 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19

WHOOPING COUGH

(PERTUSSIS)

Whooping cough is a specific infectious disease of the respiratory tract caused by a bacillus, Hemophilus pertussis, and characterized by a preliminary catarrhal stage followed by a spasmodic stage with a whooping type of cough.

Historical - First described by Baillou following an outbreak in Paris in 1578; studied by Willis and Sydenham in England 1658-1670; appeared in Germany 1724; America 1732; New Zealand 1847; Australia, 1890.

Etiology - Hemophilus pertussis discovered by Bordet and Gengou in 1906 - a small, gram negative, non-motile rod requiring hemoglobin for culture. The organism grows profusely on the cilia of the epithelial cells lining the trachea and bronchi, causing irritation and secretion of mucus. Can be cultivated from cases best by the cough-plate method, using petri plates containing potato-glycerine-agar with human blood. It has been thought by some that a virus might be associated with H. pertussis in the etiology, but this has been excluded by the production of the disease in humans and anthropoid apes, using pure cultures of the organism, and by failure to produce the disease with filtrates from such cultures.

Geographical Distribution - Endemic throughout the world except in a few places like the Faroe Islands in the North Sea, where it appears in epidemics at intervals of four to six years. It is as prevalent in warm countries as in colder climates, but causes less fatilities in warm climates because of the lower incidence of complications.

Seasonal Incidence - In most countries the disease is constantly present throughout the year with greatest incidence in winter or spring and lowest incidence in the last three months of the year. Usually the highest monthly incidence is not more than three times the lowest monthly incidence.

Race and Sex - All races are susceptible. The incidence is about equal in the two sexes but the mortality is higher in females.

Age Incidence - There is little if any inherited passive immunity in young infants. Among 10,000 cases investigated by Luttinger in New York the following age incidence and the following percentage distribution of deaths from whooping cough were found.

Age	Percentage of cases	Percentage of total whooping cough deaths
1st year	20	51
2nd year	20	27
3rd - 5th years	40	18
6th - 15th years	18	3
16 years and over	2	1

Thus 80 percent of the cases and 96 percent of the deaths occurred under five years of age. This indicates that whooping cough is primarily a disease of very early childhood and that it is much more dangerous under one year of age than at any other period. Seventy-eight per cent of adults are said to have had clinical whooping cough.

Mortality - Half of the deaths from whooping cough occur under one year of age, 78 per cent under two years of age. The general case fatality rate is less than one per cent. Accurate mortality statistics are difficult to obtain because many deaths attributable to whooping cough are reported as due to bronchopneumonia. Godfrey in New York State found that only 63 per cent of the deaths due to whooping cough were reported as such. There are probably about 10,000 deaths per year from whooping cough in the United States. The rural death rate is somewhat higher than the urban death rate, due to poorer nursing care and therefore a higher incidence of pneumonia. In New York Luttinger found a case fatality rate of 4.3 per cent among tenement families and no deaths among cases attended by private practitioners. The tenement families in which deaths occurred all had more than three children, and most of the mothers went out to work during the day.

Incubation Period and Course - Incubation period usually 7 to 10 days. Catarrhal stage lasts about 10 days, the spasmodic stage about one month with a range from one week to three months. The period of decline usually lasts three weeks, but may be indefinitely prolonged. Some cases never develop a whoop but may be infectious. This is particularly true of adults suffering from a second attack.

Period of Communicability - From the beginning of the catarrhal stage up to four weeks after the beginning of the whoop. Most infectious during the catarrhal stage with gradual diminution during the spasmodic stage. Cough plate examinations show a sharp drop in positive results after the fourth week. It is reasonably safe to release from quarantine at this time. Chronic carriers of the organism are unknown.

Complications - Bronchopneumonia is the most important. It is most frequent in the second or third week of the spasmodic stage but may occur earlier or later. Early bronchopneumonia may be due to H. pertussis itself invading the lung tissue. Later cases are caused more often by secondary invaders such as the hemolytic streptococcus or the pneumococcus. Otitis media occurs in about 7 per cent of the cases. Diarrhea is a common

complication during the summer. Gastric tetany may occur due to alkalosis from the vomiting of the acid gastric contents. Meningismus, meningitis, encephalitis, cerebral hemorrhage, emphysema (vesicular or subcutaneous) bronchiectasis, or hernia may occur. A tuberculous infection often develops into clinical tuberculosis following the disease. This should always be suspected in protracted cases of whooping cough.

Mode of Transmission-

(1) Droplet infection is the most important. The patient is dangerous within a distance of five feet. Infectiousness is greater than scarlet fever or diphtheria, less than measles or influenza.

(2) Direct contact by fingers.

(3) Fomites such as handkerchiefs, toys, drinking cups, towels, etc.

Immunity- There is little or no passive inherited immunity. One attack usually confers permanent immunity, but some adults have been proved to have a second attack by the recovery of the organism on cough plates. These cases usually do not develop the whoop, and the disease goes unrecognized but may be source of infection. This is often called "grandmother whooping cough". Some children develop immunity without a history of clinical whooping cough, probably as the result of mild infections. Complement fixing antibodies are developed during an attack and disappear after recovery. Attempts are being made to develop a skin test which will give evidence of sensitivity to the organism. The opsonocytophagic test is usually positive as immunity develops. These tests may prove to be valuable in establishing previous exposure and the immunity status of groups, and in the diagnosis of obscure cases.

Specific Immunization and Treatment - A vaccine has been developed which is represented principally in this country by the Sauer vaccine, consisting of whole killed organisms. Sauer's vaccine must be prepared from recently isolated, "smooth" strains of H. pertussis grown on media containing human blood. It contains 10 billion organisms per c. c. A total of 7 to 8 c.c. is given in three doses at weekly intervals subcutaneously as follows:

1. 0.5 to 1.0 c.c. in each arm
2. 1.5 c.c. in each arm
3. 1.5 c.c. in each arm

Severe reactions are uncommon. Complement fixing antibodies appear in the blood in three to four weeks and persist at a high level for at least twelve weeks. Smaller doses of a similar vaccine are used in Denmark where vaccination is said to be safe except in newborn infants. Vaccination should not be performed under one month of age, but should be given as soon as possible thereafter, as immunity is not developed until 3 to 4

months after inoculation. Harrison and co-workers of the United States Public Health Service have reported preliminary favorable results from the use of alum-precipitated commercial vaccine, giving one dose of 1 c.c. The results of vaccination in the prevention of whooping cough have met with conflicting results. In Denmark it is widely used and believed to be effective both in preventing the disease and in making it less severe after it occurs. Public health agencies in the United States have not adopted the vaccine as a general procedure, but it is used rather extensively in private practice.

The use of vaccine after the onset of symptoms is also believed to give favorable results in Denmark. It is not looked upon so favorably in the United States, but there are many observations in which severe cough and vomiting have been relieved following the use of vaccine. There is no evidence of the efficacy of immune horse serum for treatment of cases. Madsen states that convalescent human serum may be of some value in the early catarrhal stage. It has not been found of value as a prophylactic agent.

Prevention and Control -

(1) Prevention of infection. The following points are important:

(a) Early reporting of all cases. This is the responsibility of physicians, but since many cases are not seen by physicians, health officers should attempt to obtain reporting from all possible sources. In the presence of an epidemic, any persistent cough should be suspected of being whooping cough.

(b) Early diagnosis, preferably by the cough-plate method. This can be done in cities where laboratories are near at hand.

(c) Isolation of cases from susceptible children, especially those under five years of age. Cases or susceptibles should be removed to separate houses if possible.

(d) Placarding of houses may or may not be advisable.

(e) Avoiding crowds in the presence of an epidemic.

(f) Daily inspection of school children for catarrhal symptoms. School should not be closed. School children known to have been exposed may be excluded from the 4th to 16th day after exposure.

(g) Concurrent and terminal disinfection in the family is necessary only if young susceptibles are present.

(h) Quarantine for four weeks after onset of whoop.

(1) Vaccination.

(2) Prevention of Death.

(a) Postpone the disease until after five years of age.

(b) Careful nursing with constant attendance night and day.

(c) Avoid promiscuous contact of patient with others to avoid secondary infecting organisms which might cause pneumonia.

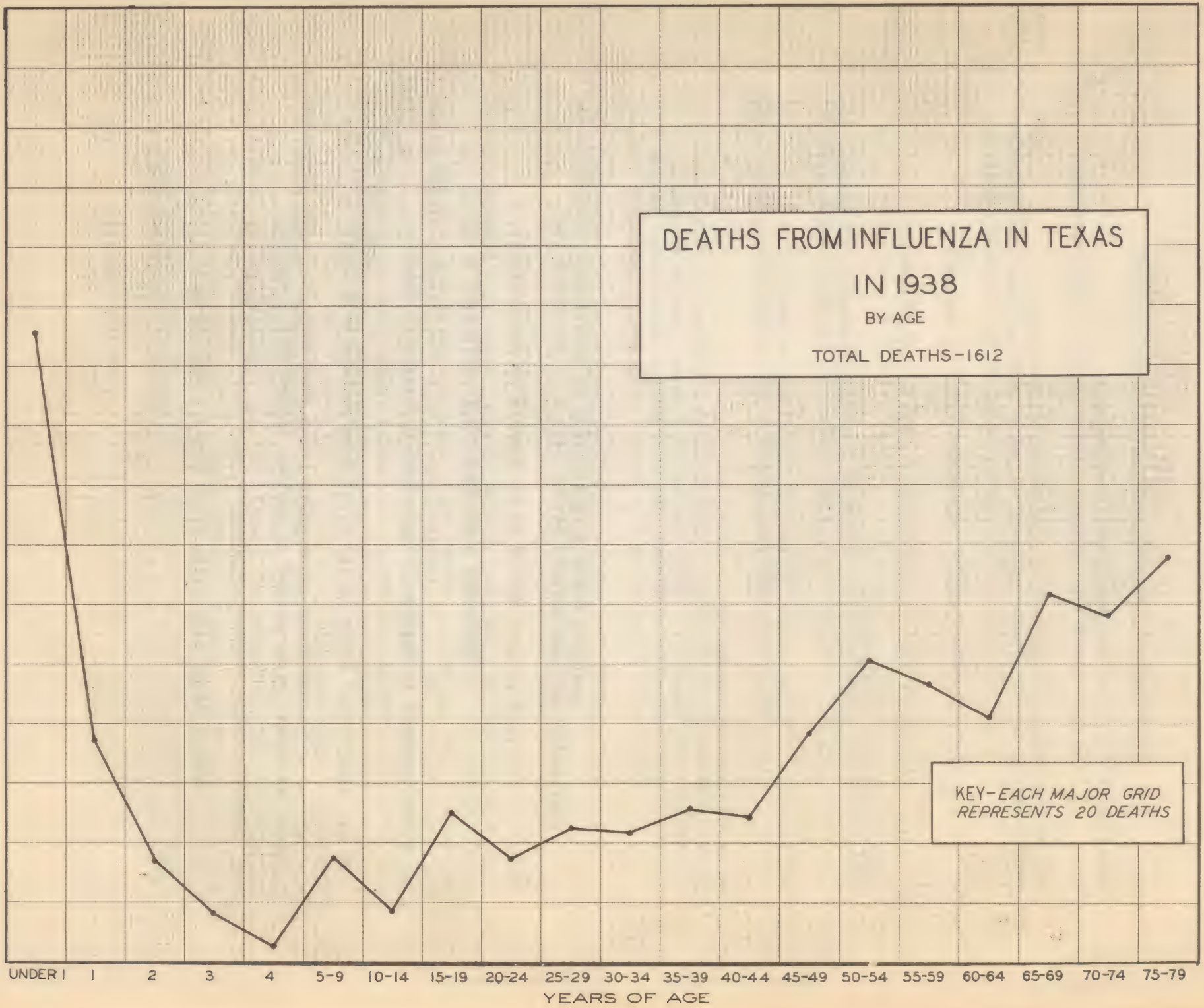
(d) In good weather keep child out of doors or in fresh air as much as possible.

(e) Keep up nutrition; counteract vomiting by frequent small meals easily digested and with little residue.

(f) Supervision of the case by a physician.

(g) Education of parents as to proper care of patient.

(h) Extension of public health nursing service to provide education and supervision of families with young children.


DEATHS FROM INFLUENZA IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-1612
KEY-EACH MAJOR GRID
REPRESENTS 20 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

INFLUENZA

1. Recognition of the disease.-- Whether occurring in a pandemic, in endemic-epidemic incidence, or as sporadic cases this disease is characterized in its typical form by sudden onset, fever of 1 to 7 day's duration, accompanied by excessive prostration, aches and pains in back and limbs, coryza and bronchitis, and not uncommonly by pneumonia as a complication. During epidemics when such cases occur in large numbers and over a wide area, other cases of less distinctive type are found to be epidemiologically related to typical cases, and in these the diagnosis would not be made without such obvious association. The clinical criteria of influenza are quite indefinite, particularly in absence of widespread prevalence of the disease. Microscopic or other laboratory procedures are of no practical value in determining or excluding the diagnosis of influenza.

2. Etiological agent. A filterable virus; associated often with various types of bacteria as secondary invaders.

3. Source of infection. -- Probably discharges from the mouth and nose of infected persons and articles freshly soiled by such discharges.

4. Mode of transmission. -- Believed to be by direct contact, by droplet infection, or by articles freshly soiled with discharges of the nose and throat of infected persons.

5. Incubation period. -- Short, usually 24 to 72 hours.

6. Period of communicability. -- Undetermined; possibly in prodromal as well as in the febrile stage and convalescent stages.

7. Susceptibility and immunity. -- Susceptibility is not general, for natural resistance or relative immunity appears to protect from one-quarter to three-quarters of persons intimately exposed to the disease even during widespread epidemics. Acquired immunity if it is actually developed by an attack of and recovery from the disease is of short duration (a few months) and of low grade, or perhaps only effective against a certain strain or strains of the virus.

8. Prevalence. -- Uncertain in pandemic, local epidemic, and sporadic occurrence, by reason of indefinite clinical symptoms. In epidemics may affect up to 50 percent of the population, especially at age groups between infancy and maturity. Commonly between December and May in North America. Occurs pandemically in cycles with intervals of several decades.

9. Method of control:

A. The infected individual, contacts and environment:

1. Recognition of the disease and reporting: By clinical symptoms only. Uncertain in interepidemic periods.

2. Isolation: During acute stage of the disease, especially in severe cases and those complicated by pneumonia.

3. Concurrent disinfection: Discharges from the nose and throat of the patient.

4. Terminal disinfection: None.

5. Quarantine: None, but visiting should be discouraged.

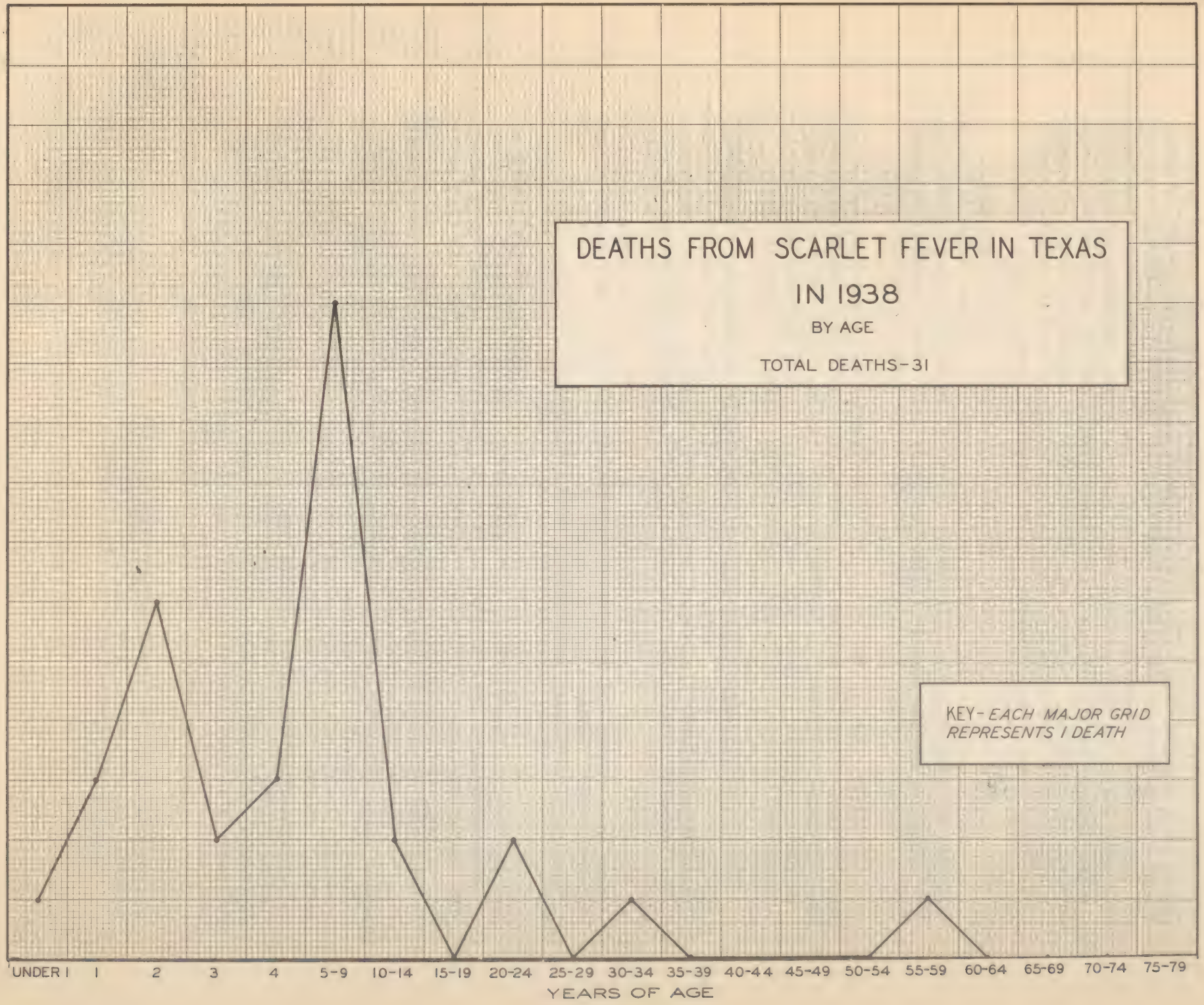
6. Immunization: None.

7. Investigation of source of infection: Of no practical value.

B. General measures:

1. During epidemics efforts should be made to reduce opportunities for direct contact infection, as in crowded halls, stores, and street cars. Kissing, the use of common towels, glasses, eating utensils, or toilet articles should be avoided. In isolated towns and institutions infection has been delayed and sometimes avoided by strict exclusion of visitors from already infected communities. The closing of the public, parochial, and private schools has not been effective in checking the spread of infection. The judicious use of masks by nurses and other attendants may prove of value in preventing infection in hospitals. Scrupulous cleanliness of dishes and utensils used in preparing and serving food in public eating places should be required, including the subjection of such articles to disinfection in hot soap suds. In groups which can be brought under daily professional inspection, the isolation of early and suspicious cases of respiratory tract inflammation, particularly when accompanied by a rise in temperature, may delay the spread of the disease. To minimize the severity of the disease, and to protect the patient from secondary infections and thus reduce mortality, patients should go to bed at the beginning of an attack, and not return to work without the approval of their physician.

2. Crowding of beds in hospitals and institutions to accommodate increased numbers of patients and other inmates is to be especially avoided. Increased spacing between beds in wards and dormitories should be carried out to reduce the risk of attack, and of the occurrence of pneumonia.


DEATHS FROM SCARLET FEVER IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-31
KEY- EACH MAJOR GRID REPRESENTS 1 DEATH
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

SCARLET FEVER

Throat infections caused by hemolytic streptococci may be divided into two general groups, (a) those associated with a scarlatinaform skin eruption and (b) those not associated with a skin eruption. There are many strains of hemolytic streptococci, some of which produce an erythrogenic soluble exotoxin.

Scarlet fever was differentiated from measles by Ingrassius in Italy in 1560. First given its name by Sydenham in England in 1665. First recorded severe epidemic occurred in New Hampshire and Massachusetts in 1735. Streptococci were first isolated from scarlet fever by Klein in 1885 and found to be present in most cases by Baginsky and Sommerfeld in 1900. Since 1920 the work of many investigators, especially Dochez and George and Gladys Dick, points almost conclusively to the hemolytic streptococcus as the cause of scarlet fever. The evidence in favor of this point of view may be summarized as follows:

(1) The hemolytic streptococcus can be recovered from nearly 100 per cent of cases of scarlet fever in the acute eruptive stage. Scarlet fever can be reproduced in susceptible persons by swabbing the throat with hemolytic streptococci.

(2) The organism produces a soluble exotoxin which, when injected intradermally into susceptible individuals, provokes a skin reaction known as the Dick test and fails in those who are immune. The toxin is neutralized in vitro by the serum of convalescent scarlet fever patients.

(3) In response to toxin injections the horse elaborates an antitoxin which neutralizes the toxin in vitro, gives the Schultz-Charlton blanching effect in scarlet fever rashes, produces a passive immunity to scarlet fever in exposed susceptibles, and has a specific therapeutic action when injected in the early stage of the disease.

(4) Injection of toxin into human subjects may produce a reaction indistinguishable from a mild attack of scarlet fever, and by suitably grading the doses of toxin, an active immunity to scarlet fever is induced.

Incidence

Scarlet fever is most prevalent in the temperate zone, especially in colder regions, but not in the Artic zone. It is primarily a cold weather disease, but epidemics may extend through the summer. Incidence has remained about the same for the past forty years in the United States, higher rates occurring irregularly every few years. The seasons have no effect upon its virulence, i.e., the case fatality rate is about the same the year round. In hot climates scarlet fever is rare, and hemolytic streptococci are rarely found in throat cultures. The disease is milder in the southern than in the northern part of the United States.

Season - In United States, maximum incidence for northern states in January, for southern states October to November; minimum for northern states August, for southern states June to July.

Age - Incidence low in first year, rapidly increases up to sixth year, then falls more gradually to twentieth year, after which it remains very low.

Susceptibility - Zingher, in New York, demonstrated that susceptibility to scarlet fever as determined by the Dick test increased from the fifth month of life onward and was greatest in the one to two-year period when over 70 per cent of children were susceptible. In the age group over 20 years the percentage of susceptibles was reduced to about 18 per cent. He also found marked differences in susceptibility according to social status, children in the better class of homes yielding a susceptibility rate of 83 per cent as against a rate of 22 per cent among children from poor homes. Urban groups are less susceptible than rural groups. Groups in institutions are usually less susceptible than the general population.

Sex and Race- Slight excess of males up to six years. Marked excess of females above seven years. This is probably due to greater exposure of females in higher age groups because they nurse the cases. The incidence among whites is three times as great as among negroes. This is due in all probability to missed and unreported cases in negroes, but there may be a relative racial immunity.

Mortality - has fallen in the past 40 years from 40 to 2 per 100,000. Fifty-two per cent of deaths occur under the age of 5 years and 80 per cent occur under the age of 10 years. For all ages the case fatality is 20 per cent higher among males.

Case fatality -(i.e., percentage of fatal cases among total cases) has gradually been falling for the past 40 years in most of Europe and in America (Sweden 1880, 17 per cent; 1920, 1.9 per cent). The case fatality rate averages about 2 per cent at the present time and is higher for the age periods under five years. In Russia, Poland, Bulgaria, Hungary, Italy, and China the fatality has remained higher. In Japan it is decreasing but is still higher than in America. It appears that the case fatality in general is lowest in places where quarantine is best carried out. Chapin (1926) believes that this is because the more virulent strains are being weeded out by quarantine. He believes that strains which are usually less virulent may occasionally increase in virulence, but that in well supervised communities they are soon eliminated. The non-virulent strains persist because of mild and missed cases. The mortality rate averages 2-3 per 100,000, which is lower than the rate of other acute infections.

Incubation period - One to seven days, usually two to five days.

Modes of Transmission - The organisms causing scarlet fever usually enter and leave the body through the mouth and nose. The discharges from draining sinuses and abscesses also contain the infectious agent. Streptococci may also enter the body through wounds and burns (surgical scarlet fever), and the uterus during the puerperium. The human is the only source of scarlet fever. The disease may be transmitted either by:

(1) Direct contact, by droplet infection, from typical or atypical cases of the disease. The clinical manifestations of scarlet fever are more varied than those of any other communicable disease. All degrees of severity of scarlet fever may be seen from mild upper respiratory infections or sore throat without a rash to fulminating eptic or hemorrhagic cases.

(2) Indirect contact with contaminated articles, linen, hands, infectious discharges, and food, especially milk. It is probable that the hemolytic streptococcus of scarlet fever can live longer outside the body of the host on fomites than most organisms. Milk becomes infected from human sources, usually a milk handler excreting hemolytic streptococci, either in convalescence from scarlet fever or as a mild or missed case. The cow's udder may become infected and act as a culture medium. Many extensive milk-borne epidemics of scarlet fever have occurred. The occurrence of a number of cases of scarlet fever almost simultaneously in a community usually indicates that milk is the source of infection. Milk-borne epidemics of scarlet fever may occur at any time during the year.

"Return cases" are those occurring in a patient's family soon after his recovery. They are more frequent after isolation periods under three weeks, but even after 9 weeks' isolation one per cent give rise to return cases.

Period of Communicability and Carrier Problem - Depends entirely upon the presence of streptococci in discharges of patient. Nicholls (1927) cultured throats of 40 cases at end of 3 weeks and of six cases at the end of four weeks and found all of them to harbor S. scarlatinae. Two out of three still harbored the organism at the end of five weeks. Gordon (1927) in a series of cases discharged on the twenty-first day or later found that 64 per cent of cases not treated with antitoxic serum still harbored hemolytic streptococci, while only 38 per cent of treated cases harbored them. Of the severe untreated cases 68 per cent were positive on discharge; of the mild untreated cases 11 per cent were positive.

Gordon states that the number of infecting cases varies inversely with the age of the patients; that is because of the fact that young children are more likely to develop one or more complications and continue to excrete the organisms, usually the throat, ears, suppurating glands, nose, or sinuses. A nasal discharge is especially dangerous and is least suspected of being infectious. A "return case" contracted from one of mild type is frequently characterized by unusual severity, and vice versa.

Recognition of the fact that carriers of scarlet fever streptococci exists affords an explanation of recurrent outbreaks in schools and institutions, and sporadic cases with no known scarletinal contact. Milk-borne epidemics sometimes appear to have been started by carriers. There are so many strains of hemolytic streptococci that the detection of carriers of the scarlet fever strain is more difficult than diphtheria, typhoid, or meningococcus infections. Both convalescent and contact carriers may be present in a community. Carriers of more than three months' duration are rare. Carriers of scarletinal streptococci usually have a negative Dick

test so that an immune person may harbor these organisms. Jones has found that when the prevalence of scarlet fever increases the percentage of healthy carriers increases greatly. This is similar to what has been found in connection with the meningococcus. The increase of cases is the result of widespread dissemination of the organisms.

The handling of carriers is a difficult problem. On discharge from the hospital, as a matter of precaution, it is best to advise patients to spend a week or two in the open air, to avoid intimate contact, especially with children, and to regard any recurrence of nasal discharge or catarrhal secretions from the upper respiratory tract as dangerous.

Immunity - One attack of scarlet fever usually produces a permanent immunity. Second attacks occur in about one per cent of the individuals who have had scarlet fever. In one series of 5,000 cases there were 25 relapses, 15 of which occurred within 30-35 days of the original attack.

Control and Prevention

A. General Measures:

1. Early diagnosis - Often difficult due to absence of rash. In differential diagnosis of scarlet fever, the following diseases should be kept in mind: rubeola, rubella, diphtheria, exfoliative dermatitis, food, serum, and drug rashes. The Schultz-Charlton skin blanching test is useful when the rash is present. It consists of an injection of 0.1 - 0.2 c.c. scarlet fever antitoxin or convalescent serum intradermally into a portion of the body where the eruption is distinct (usually the abdomen). If the test is positive, there will be an area of blanching about 4 cm. in diameter. Blake reports that in undoubted cases of scarlet fever blanching occurred in 80 per cent and that a positive test occurs more frequently when the rash is diffuse rather than punctuate. He states that the average interval between the test and blanching was fourteen and a half hours, the shortest being six and a half hours. It may be as long as thirty-six hours. The absence of blanching does not exclude the diagnosis if characteristic symptoms and signs are present.

The Dick test becomes negative during convalescence in a majority of cases, though in some instances it remains positive indefinitely.

It is difficult to determine with certainty by bacteriological methods the particular hemolytic streptococcus of scarlet fever. Nevertheless throat cultures should be taken. The presence of such organisms in relatively large numbers in pure culture gives support to the diagnosis of scarlet fever and helps to rule out the possibility of diphtheria.

(2) Notification - This is required immediately and on weekly report cards. Investigation of cases by the local health officer to determine the source of infection and contacts. Placarding is advisable.

(3) Isolation - Hospitalization is advisable in severe cases and where the case cannot be properly isolated at home. It is also advisable where there are many young children in the home. It is unnecessary in milder cases where proper isolation can be provided at home and where there is good cooperation between the medical attendant and the health authorities.

(4) Quarantine of contacts - Exclusion of food handlers from work until seven days have elapsed since last exposure to a recognized case. Swabbing of the throats of contacts may be done to determine the presence of hemolytic streptococci. No one except the nurse or attendant should come in contact with the patient. Adults, not in association with the patient, whose occupation does not bring them in contact with children or the handling of milk or food may be given a permit to enter and leave the premises. Children who are not immune as shown by the Dick test may be removed and quarantined in another household where there are no children for a period of seven days and then released and may return to school, but may not return to their homes until the patient has been released from isolation. Susceptible children who remain in the house with a case must stay in quarantine until the patient is released from isolation.

(5) Concurrent Disinfection - All articles which have been in contact with a patient and all articles soiled with discharges from the patient should be destroyed or sterilized.

(6) Release - Three to four weeks from the onset of the disease is long enough in uncomplicated cases. It is no longer considered that the scales from the desquamating skin contain the infectious agent. Patients should not be released if there is an complication, suppurative process, or nasal discharge. Since post-scarletinal nephritis may occur during the third or fourth week, it is well to keep the patient in bed or restricted for four weeks, and isolation is the simplest means of encouraging this. Three negative throat and nose cultures for hemolytic streptococci before release should be of advantage in minimizing return cases, but this is practicable only in hospitals. Even there it is not practicable to identify the organism as a scarlet fever strain. In cases having a complicating discharge, however, three negative cultures should be required for release from quarantine.

(7) Terminal Disinfection - Thorough cleaning of the room and its contents is required.

(8) Supervision of milk supply - Careful supervision of the production of raw milk supplies is essential but will not prevent epidemics. Pasteurization of milk is the only safe preventive measure.

B. Specific Measures

(1) The Dick test - This test determines susceptibility to scarlet fever. It is performed by the intradermal injection of 0.1 c.c. of the toxin of the S. scarletinae grown form 4 - 6 days in a broth culture. The amount

of toxin injected is known as one skin test dose (S.T.D.): that is, the smallest amount toxin which will produce a positive Dick reaction in known susceptible individuals and a negative reaction in immunes.

The toxin is usually injected on the flexor aspect of the forearm and must be read within 24 hours. A positive Dick test consists of an area of erythema 10 mm. or more in any diameter at the site of injection. There is usually no swelling or induration associated with a positive reaction as is found in a positive Schick test. The Dicks state that there is some correlation between the intenseness and size of the reaction with the degree of susceptibility of the patient.

Pseudo reactions resulting from sensitization to the protein in the test material may occur, especially in older children and adults. A control test should be made in dealing with the above groups (older children and adults) with the toxin heated for a long period as it is heat stable.

The Dicks claim a high degree of precision for this test and report one group of 22,856 Dick negatives who have passed through one or more epidemics of scarlet fever without contracting the disease. They also report another group of 2,557 nurses and interns with negative reactions who were constantly exposed to scarlet fever and remained immune. Others, however, have occasionally encountered scarlet fever in supposedly immune persons. The test is of practical value in institutions for children where immunization is contemplated, and in families where a case of scarlet fever has occurred.

(2) Immunization - (a) Active. This consists of the subcutaneous injection of scarlet fever streptococcus toxin in five increasing doses at weekly intervals, namely, 500, 2,000, 8,000, and 80,000 S.T.D. The patient should be given a Dick test one month after the last injection. If the test is negative the patient is probably immunized. If the test is positive, an additional dose of 90,000 S.T.D. should be given and a retest done one month later. This series of injections is usually without danger but severe, unpleasant local and general reactions, including erythematous rashes, occur occasionally. The Dicks claim that with this method 95 per cent of susceptibles will become Dick negative within two weeks and 90 per cent will remain immune for one to five years.

The development of a toxoid comparable to the diphtheria toxoid has not yet been accomplished. Veldee of the U. S. Public Health Service has separated the erythrogenic factor of the toxin and precipitated it with tannic acid. Preliminary tests indicate that this substance injected intradermally will cause the Dick test to become negative, and that it rarely produces severe reactions.

Due to the fact that scarlet fever is generally a mild disease with low case fatality, it is not considered advisable to advocate active immunization against scarlet fever in the general population. There are,

however, certain circumstances under which this procedure may be used with benefit. They are:

(1) in epidemics

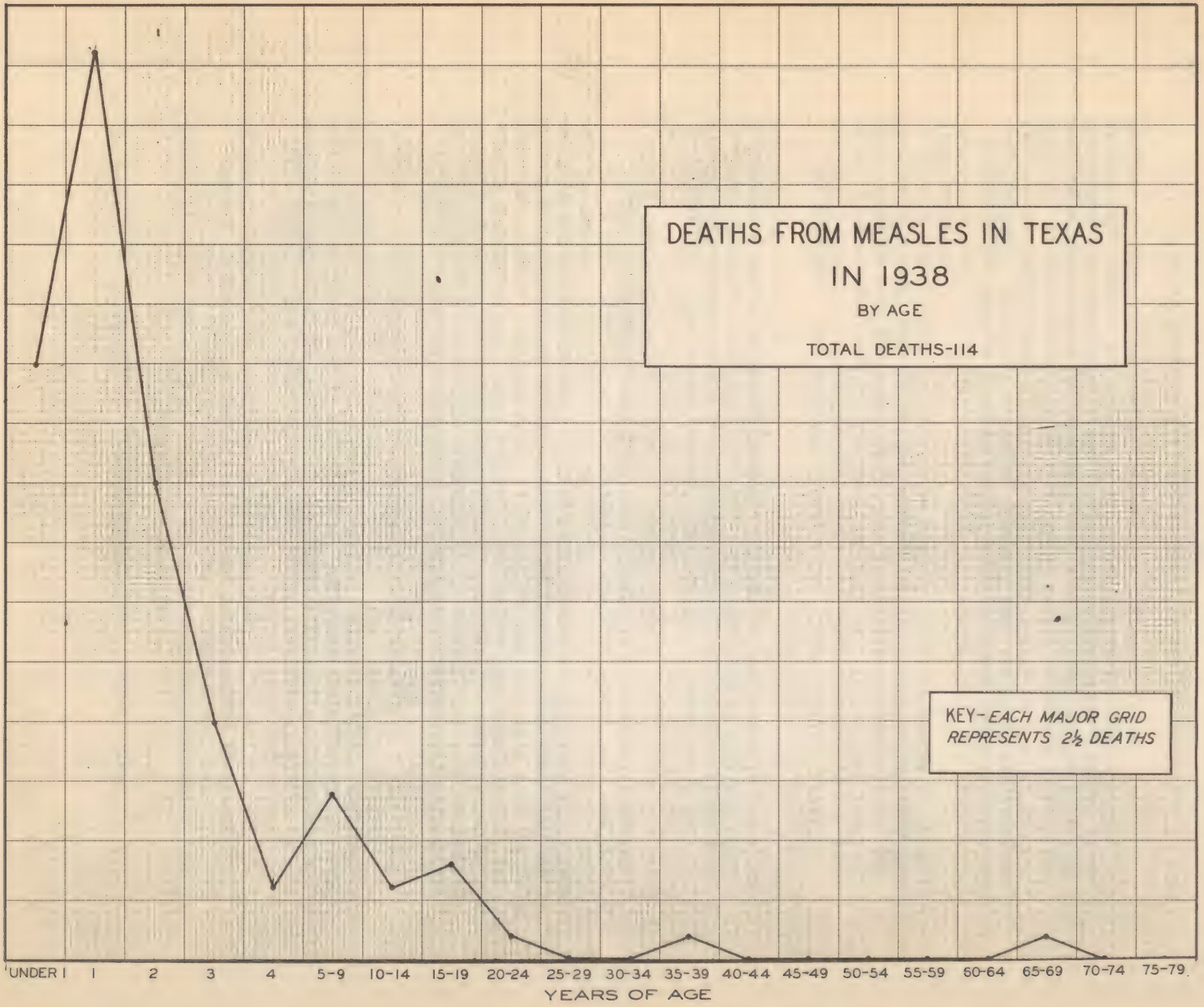
(2) in certain family groups

(3) in institutions caring for large groups of children, and

(4) in personnel of hospitals.

(b) Passive. Scarlet fever antitoxin or convalescent serum can be employed for this purpose in contacts where immediate protection is desired. The usual practice is to inject 5 c.c. of scarlet fever antitoxin subcutaneously or intramuscularly. This gives immediate protection in a large per cent of cases but the duration of immunity is short, probably no longer than two weeks. A dose of 10 c.c. of serum will probably protect for four weeks, but one should not rely on this as cases of the disease have been reported from six to seven days after this procedure. The production of passive immunity is of value under circumstances where there are cases under poor hygienic conditions, and where a number of small children are exposed and where isolation cannot be carried out. It is not advocated generally. If convalescent serum is used, the dosage should be increased to two to three times that of the antitoxin.

Since the advent of sulfanilamide the antitoxin and convalescent serum are usually reserved for the most severe type of case. While highly effective in treatment of scarlet fever, both are expensive, and the antitoxin often gives very unpleasant serum reactions.


DEATHS FROM MEASLES IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-114
KEY- EACH MAJOR GRID
REPRESENTS 2½ DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

MEASLES

(RUBEOLA - MORBILLI)

Measles is a specific infectious disease characterized by fever, catarrhal symptoms, an exanthem and desquamation.

Historical - The first definite mention of measles was made by an Arabian physician about 900 A.D. The disease was at first considered a variety of smallpox, and it was not until the seventeenth century that it was recognized as a distinct clinical entity. Much of our present knowledge is due to the work of Panum who studied the disease in the Faroe Islands in 1846, where it had not existed for 65 years.

Etiology - The etiological agent at the present time is considered to be a filterable virus. Experimental work, however, has been carried on without the specific causative agent being discovered. Innoculation of animals and humans with blood and nasal secretions from patients have produced the disease. Various bacilli, cocci, and protozoa have been indicated. The virus is present in the blood and nasopharyngeal secretions from one to two days before rash appears. Virulence increases as incidence of the disease increases.

Immunity - Natural immunity is rare. There is a universal susceptibility to the disease.

Prevalence - The disease has universal distribution. It may be endemic, but epidemics tend to occur every two years with the appearance of large numbers of susceptible people. It ranks next to smallpox and influenza in degree of contagiousness. Epidemics are common in urban communities.

Age Incidence - All ages are susceptible. Intra-uterine infection has been reported. It has been estimated that approximately 90 per cent of all persons surviving to the twentieth year of life have had measles. The disease is most prevalent in the age groups five to nine and ten to fourteen; 75 to 80 per cent of cases occur before the fifth year but this is probably not correct as such cases are not as well reported as those of older children.

Mortality - Measles is rarely fatal per se. Death is usually due to complications. The total mortality reported from the U. S. Registration Area varies roughly from 2,500 to 8,500 deaths yearly or 5 to 10 per 100,000. A fatal outcome in measles is due mainly to complications, the most important of which is pneumonia, since approximately 95 per cent of the deaths associated with measles results from pneumonia. The relationship between measles and pneumonia was shown during the World War by Vaughan who made the following statement in regard to this:

"(1) Of every thousand men with measles, 44 had pneumonia and 19 died.

(2) Of every thousand men without measles, 17 had pneumonia and 2 died.

(3) A person who has recently had measles is ten times more likely to die from pneumonia than is the person who has not recently had measles."

In considering the question of mortality from measles, superimposed infection should be mentioned as another factor which produces death. The possibility that a tuberculous focus may be awakened to activity as a result of measles should always be considered as a remote cause of mortality from measles. There is no sex predisposition.

Measles is a disease that should not be hospitalized except in case of actual complication. Experience has shown that the incidence of complications is greatly increased where measles cases are uniformly sent to isolation hospitals. This is probably due to interchange of pathogenic organisms, or places the individual in contact with organisms which are not natural flora in his own respiratory system.

Case Fatality Rate - Fatality rates for all persons attacked are probably not over 3 or 4 per thousand cases. It varies greatly with age, physical condition and social and environmental conditions. Fatality rates up to 35 per cent have been noted in infant asylum outbreaks, due to the enhancement of the usual risk in young children by the prevalence of rickets and other forms of malnutrition, the overcrowding and inadequate nursing and hygienic care. In whatever degree these factors operate in community life, they serve to increase the fatality rates. The fatality rates by age probably approximate 30 - 50 per thousand cases in children under one year old; 15 - 30 per thousand at age one; 10 - 15 at age two. Between five and fourteen the fatality rate is probably not more than 2 per thousand, gradually increasing in adult life to approach in old age the rates of early childhood.

Racial Distribution - Measles attacks all races.

Seasonal Incidence - Measles is more common in winter and spring. Incidence increases in cold weather. In the U. S. the greatest prevalence is from March to June.

Incubation Period - 7-14 days. Rash appears from 13-14 days after infaction. When convalescent serum has been used, the incubation period may be prolonged, in some instances up to 21 days.

Period of Communicability -From onset of catarrhal symptoms to the disappearance of the eruption. It is infectious during pre-eruptive stage. Five days before and five after eruption appears is the most highly infectious period of the disease.

Mode of Transmission

(1) Close proximity and direct contact is the most important. The infectious agent is disseminated by minute droplets given off during coughing or sneezing.

(2) Indirect contact. Short exposure during period of communicability will suffice for transmission. A carrier state is not known to exist.

Control Measures -

(1) Early diagnosis - Koplik's spots - rise in temperature - edema of conjunctiva - catarrhal symptoms.

(2) Isolation of cases and contacts for 16 days should be practiced though only 7 to 10 per cent of the population will be protected by this procedure. Sir Arthur Newsholme believes "that the measles problem is very similar to the influenza problem. In both, so far as our present knowledge goes, the chief hope of saving life lies in the preventing or in diminishing the mortality from streptococcal or pneumococcal pneumonia in the disease." The primary benefit resulting from isolation of patients is to reduce promiscuous contact and likelihood of secondary infection. In hospitals and institutions both uncomplicated and complicated cases should be strictly isolated. If measles appears in an institution which cares for young children or if measles is present in a community where such an institution is present, quarantine of the institution may be of some value in preventing the introduction of measles.

(3) Concurrent and terminal disinfection.

(4) Schools should not be closed.

(5) Education of public as to the seriousness of the disease.

(6) Prompt and complete reporting of cases.

(7) Care of the eyes.

(8) Special measures should be designed primarily to reduce mortality. Infants in asylums should be kept in best possible condition, special attention being given to prevention and cure of rickets. Such institutions should in effect be quarantined during outbreaks in vicinity, and if invaded, special attention should be given to care of those attacked. Convalescent serum, should be given, if available, to children under three, but should not be relied upon alone. With a potent serum the disease will usually either be modified or prevented. In the community at large, extra nursing personnel should be employed. Cases should be investigated to ascertain family contacts under three years of age so that the parents may be instructed in the particular

danger of measles to these children and shown how best to arrange for the care of the sick and those who will probably come down later. Families in the neighborhood who have children of this age, or physically handicapped children, should also be visited and instructed, and efforts made to improve physical condition if impaired. Convalescent serum should be made available for use in suitable cases. Adequate medical and nursing care should be provided for the indigent, and in exceptional circumstances hospitalization should be provided. Cases should be isolated for their own protection.

Proper rest during and after an attack is of great importance in preventing sequelae. The patient should go to bed on the eighth day after exposure and remain in bed until all dangers for complications are past. The prevention of tuberculosis following an attack of measles is one of the most important points to be emphasized.

Prevention - Exposed people can be passively immunized for two to four weeks with convalescent serum taken from a person who has had measles preferably six to ten days after recovery. Convalescent serum may be taken as long as four months after recovery. A dosage of 5-10 c.c. may prevent disease or cause the disease to be milder. Its use is particularly desirable in very young or debilitated children.

The following is a brief review of the literature on the prophylactic use of immune serum:

1. In 1915, Doctor Charles Herman, New York City, rubbed nasal secretions from patients on nasal mucous membrane of non-immune children. Produced modified disease.

2. First successful immunization with convalescent serum done by Nicolle and Conseil in 1916.

3. Von Torday immunized 2,000 exposed children - only 3 per cent were not protected to some extent.

4. Debre and Ravina injected convalescent serum from 7-10 days after exposure; complete protection not brought about but produced mild disease as evidenced by prolonged incubation period; suppression of invasion symptoms; slight fever; absence of catarrhal symptoms and Koplik's spots, faint eruption; short course; mild or no constitutional symptoms.

5. Degkwitz was first to use whole adult blood of donors who had had measles. Reitschel and Zingher independently used serum or whole blood from immune adults and found that some protection was derived from this method but that dosage had to be increased.

6. Optimum time for obtaining convalescent serum is 6 to 10 days after patient's temperature has reached normal.

7. Convalescent serum can be kept as long as six months.

8. Dosage for complete protection. Children up to 3 years of age not exposed more than 4 days - 2.5 c.c.; if exposed from 5 to 6 days - 5 c.c.; 7.5 c.c.; if exposed for 7 days; larger doses for older children. After 8th day of exposure, disease may not be prevented by infection of immune serum but may be modified. Whole blood instead of serum may be used in double the above dose.

9. To cause modified measles, Zingher advocates the following methods from fifth to tenth day after exposure; 2.5 c.c. of convalescent serum; 5 c.c. serum from brother or sister who has had the disease or 10 c.c. of adult immune serum.

10. Convalescent serum or adult whole blood is not effective when used therapeutically.

11. Use of prophylactic serum indicated in

(a) children in first four years of life,

(b) children suspected of having tuberculosis,

(c) children who have other contagious diseases, especially in hospitals and in other institutions, and

(d) in other instances where the health, age and environmental circumstances make the disease dangerous.

McKhann and Coady have reported on the possibilities of preparing extracts of the placenta which might be used in protecting infants and children against certain diseases. These workers made a two per cent sodium chloride extract of several placentas and precipitated the globulins with ammonium sulphate. The ammonium sulphate was removed by dialysis. The solution which remained after this procedure was passed through a Berkfeld filter and upon injection into animals was found not to produce any harmful effect upon the tissues. Upon qualitative determination, such a placental extract:

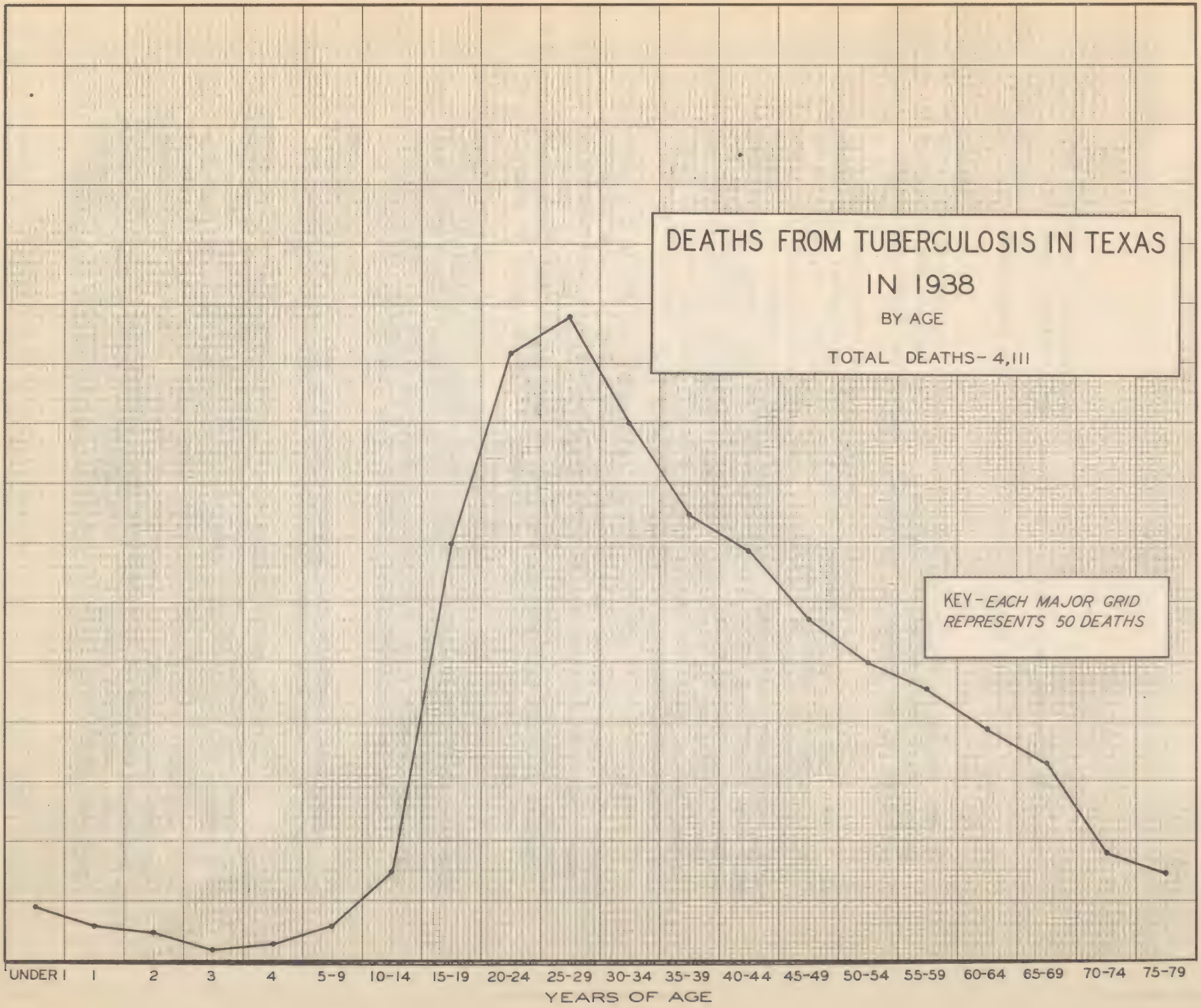
(1) neutralized diphtheria toxin,

(2) blanched scarlet fever rashes,

(3) neutralized poliomyelitis virus, and

(4) prevented measles in exposed susceptible patients.

They have reported results with this extract comparable with those obtained with human convalescent serum both in the prevention and modification of the clinical attack of measles. This work is well confirmed, and will be of great value in producing passive immunity against measles and modifying the disease under suitable circumstances and it will be possible to have sufficient quantities of the material for general use.


DEATHS FROM TUBERCULOSIS IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS- 4,111
KEY- EACH MAJOR GRID
REPRESENTS 50 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

Evidence of past or present infection in the absence of clinical symptoms can be determined by a variety of specific tuberculin reactions, among which the Mantoux intradermal test is the most reliable. In the presence of early constitutional symptoms with or without pulmonary signs, the existence or location of pulmonary or other thoracic lesions can best be revealed by X-ray. When fever, cough, loss of appetite and weight and physical signs or ausculation and percussion are found, the pulmonary lesion is already well developed. Discovery of the tubercle bacille in the sputum confirms the diagnosis not infrequently in early cases but is an evidence usually of a well advanced lesion.

Pulmonary tuberculosis is caused by the human type tubercle bacilli, although the bovine type has been isolated from pulmonary lesions in man.

Source of infection. -- The specific micro-organism present in the discharges, or articles freshly soiled from the discharges from any open tuberculosis lesions, the most important discharge being sputum. Of less importance are discharges from the intestinal and genito-urinary tracts or from lesions of the lymph nodes, bone and skin.

Mode of transmission. -- Usually through the discharges of the respiratory tract, by direct or indirect contact with infected persons, by means of coughing, sneezing or droplet infection, by kissing, by use of contaminated eating or cooking utensils and possibly by contaminated flies and dust. Infection rarely results from casual contact but usually results from continued exposure.

During the year 1938 the greatest number of deaths from tuberculosis in Texas occurred in young adults in the age group between twenty and thirty in both males and females. Comparison of the figures for the last few years show that there has been little change in the annual tuberculosis death rate as compared with the steady decline of many other states. The logical conclusion is that our position is stationary, and until our present plans have been fortified a plateau trend of rates is to be expected. A plan of attack was so well brought out by Dr. J. A. Myers, in a recent nine point control program for the United States, that it can be well repeated and applied to Texas.

"The following procedures," he states, "directed by the medical profession and carried out by its members in cooperation with closely allied groups and an informed public should suffice to control the disease:

"1. Administer the tuberculin test to every one.

"2. Make X-rays of the chests of all positive reactors approaching and beyond adolescence.

"3. Make complete examinations of all whose X-rays present shadows which might be due to tuberculosis in order to arrive at the true diagnosis.

"4. Arrange for an adequate number of beds in private and public hospitals and sanatoriums so that all persons who have tubercle bacilli in their sputums which cannot be eliminated quickly can at once be removed from their homes.

"5. Arrange to treat or keep under close observation all who have tuberculous lesions, the progressiveness or activity of which cannot be determined at once. The physician can manage the disease of the majority of such persons in the home.

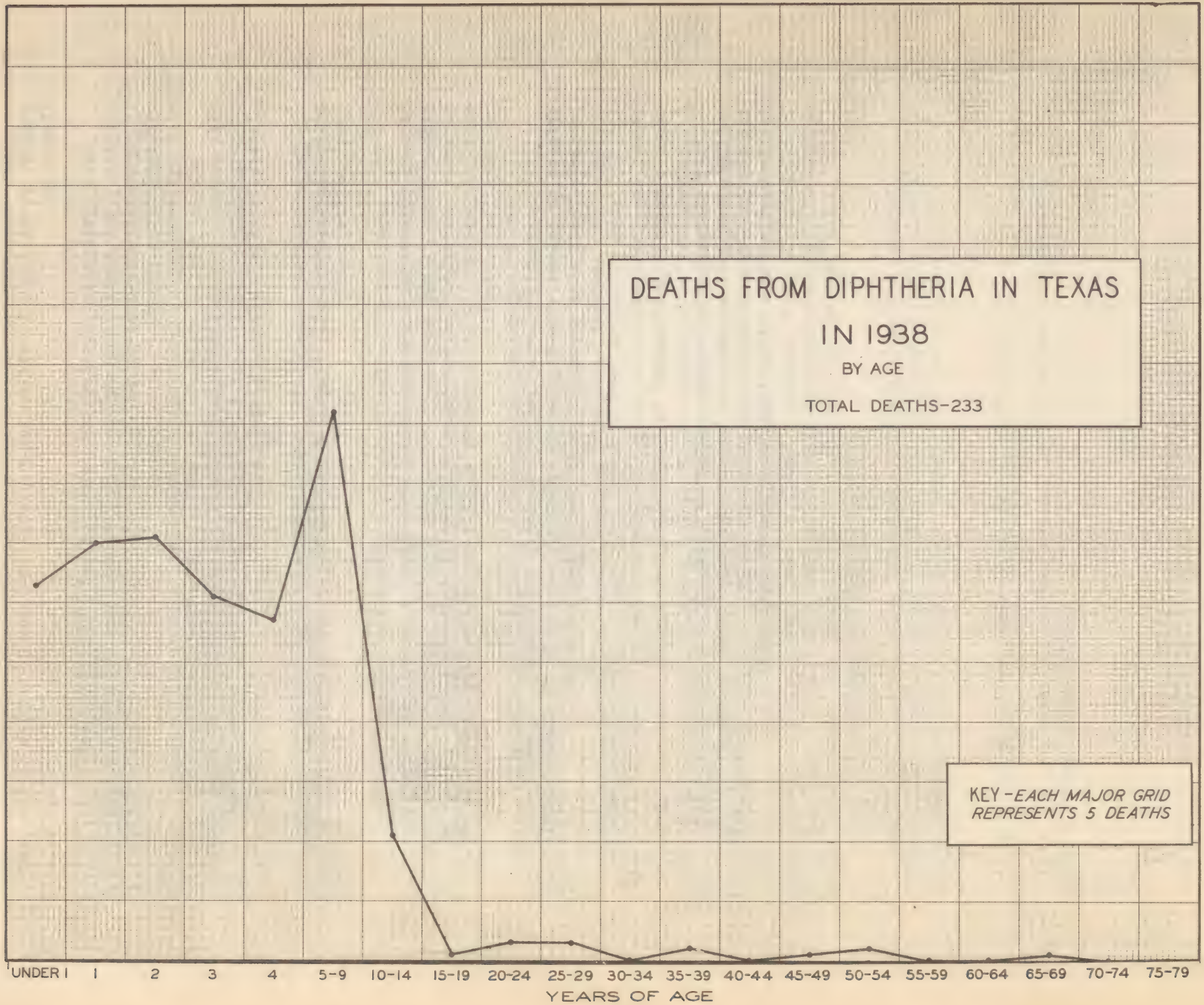
"6. Since tuberculosis is usually arrested, not cured, and is a relapsing disease, all persons whose disease is under control should be examined frequently.

"7. Arrange for the protection of citizens against the importation of persons with communicable tuberculosis by providing adequate examinations of all persons entering the country's ports and crossing its borders. Exercise the same precaustions for those of other nations by examining adequately this country's citizens before they leave its ports or cross its borders.

"8. Support the veterinarians at every opportunity in their efforts to control the disease in animals, because it is transmissible to man. Their past accomplishments merit this and more.

"9. Take an active part in local and national tuberculosis associations, the local medical society, the American Medical Association and all other organizations engaged in tuberculosis work."

Any tuberculosis program suggested would contain many of the preceding nine points. They are not necessarily listed in the order of importance. Any difference in opinion will be in how far these points can be carried out rather than their theoretical value.


DEATHS FROM DIPHTHERIA IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-233
KEY - EACH MAJOR GRID REPRESENTS 5 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

DIPHTHERIA

Diphtheria is an acute infectious disease caused by the Klebs-Loeffler bacillus characterized pathologically by the formation of a pseudomembrane on some mucous surface, usually the oropharynx or nasopharynx. The surface of wounds may also become infected. A soluble toxin is elaborated which is absorbed and to which the symptoms and lesions of the disease are due. The bacillus itself may find its way into the blood and tissues, but the essential factor in causing clinical manifestations of the disease is the soluble toxin. One attack does not give permanent immunity.

Historical note. - Although it is a little more than one hundred years since diphtheria received its present name, the history of the disease can be traced to a remote period. It is doubtful if diphtheria was known in the time of Hippocrates, though certain passages imputed to be his writings suggest that it was recognized at that time. It was not until the first century A. D. that an unmistakable description of diphtheria was given by Aretaeus of Cappadocia under the name of Egyptian or Syriac Ulcer, owing to its having originated in Egypt and Syria, thence spreading to all European countries. In 1883, Klebs described for the first time the bacillus now known to be the specific organism, and which was first cultivated in 1884 by Loeffler, hence the name Klebs-Loeffler bacillus. The work of Loeffler was confirmed by Roux and Yersin, who were not only able to produce paralysis in animals by inoculating them in the trachea and also subcutaneously with diphtheria bacilli, but also demonstrated the existence of a diphtheritic toxin. The toxin was obtained by filtering the culture fluid through porcelain. The discovery of diphtheria antitoxin was made in 1890 by Behrin and its practical value shown by Emile Roux in 1894. The test of immunity to diphtheria was introduced by Schick of Vienna in 1914. The use of toxin-antitoxin for immunization purposes, suggested by Theobold Smith in 1907, was first applied clinically by Behring in 1913. Its employment, however, on a large scale was due to the work of Park and Zingher (1917-1918) in the United States.

Prevalence or Incidence of Diphtheria - Diphtheria is most prevalent during childhood but no age is exempt. The incidence of the disease is at least during the first year of life, and higherst from five to seven years of age. The greatest susceptibility to diphtheria is in the age group from one to five, and about 80 per cent of the deaths occur under five years of age. In the Death Registration Area of the United States in 1900 diphtheria death rate was 43.3 per 100,000. In 1932 the rate was 4.5. Diphtheria is essentially an endemic disease, although it may become epidemic. The periodicity of diphtheria is indefinite.

Causative Organism. - Diphtheria is due to the invasion of specific microorganism, the bacillus diphtheriae (Klebs-Loeffler). The bacillus grows locally on mucous surfaces or on the skin. When kept in a dry state, under certain circumstances, the bacillus possesses considerable resistance. A culture may be obtained from a dry membrane under certain conditions. The organism is very resistant to cold but is rapidly killed by heat, especially by the action of the sun's rays. It may be also readily destroyed by

antiseptics (phenol, $HgCl_2$). It grows best on Loeffler's medium at a temperature of 37° C. The dissemination of the bacillus in different parts of the body is rare and when so found is probably not of much pathological significance. The bacilli begin to disappear from the site of the lesions as the acute symptoms subside so that by the end of a month about 85 per cent of diphtheria patients are free from bacilli and by the end of the second month usually 98 per cent; the remainder may become chronic carriers. Some authorities maintain that the diminution of virulence precedes the disappearance of the organism, but most investigators maintain that they retain their virulence throughout the course of the disease.

Modes of Transmission. - Period of incubation may be from two to seven days, but the average is from three to five. The attack rate - on the average about 6.5 per cent of those exposed develop clinical diphtheria. The infective agent usually has its exit from the body in the secretions of the mouth and nose, and it also gains admission commonly through the mouth and nose. The source of infection in the spread of the disease is the case or carrier. The case may be typical, atypical, or mild. The "missed" case is potentially very dangerous. The infection is usually transmitted directly from one person to another, as by kissing or droplet infection in coughing, sneezing, and speaking. The bacillus may also be conveyed indirectly from one person to another in many ways, as for example, toys, pencils, food, fingers, spoons, cups, handkerchiefs, or any object on which may be found fresh secretion from a case or carrier. The latter mode of transmission is most common among children. Personal hygiene should therefore be emphasized as an important factor in the prevention of diphtheria. This is not a disease that is easily transmissible and is, therefore, more commonly spread from one person to another by direct contact. Experience clearly teaches that diphtheria is spread mainly by typical and atypical (abortive) cases, recent convalescents, mild and missed cases and carriers. Occasionally milk becomes contaminated, resulting in the spread of diphtheria. It is claimed that domestic animals, especially pets, may transmit the infection to a human being under exceptional circumstances.

Diphtheria Carriers. - It is generally accepted that diphtheria continues as a disease entity by means of chronic carriers who act as sources or reservoirs of infection and from these the organisms are spread under favorable conditions to susceptible individuals. It is conceded that diphtheria would not be much of a public health problem if it were not for "carriers". A possible carrier can be diagnosed without difficulty and the virulency of the organism found can be determined by laboratory tests. However, the treatment of the carrier offers the most difficult phase of the problem.

Types of Carriers: -

1. Incubationary. These may play some part in the dissemination of the infection, but the incubation period of diphtheria is so short that this would not be an important factor in its spread.

2. Convalescent carriers. Virulence tests are not necessary in such carriers because it has been found that the organisms in 90 per cent of canvalescent carriers are virulent. Physical defects of the nose and throat predispose to the chronic carrier state.

3. Contact carriers. These occur among attendants, members of the family and contacts of cases and carriers in from 10 to 20 per cent of exposures. The organisms are virulent in 80 per cent of the contact carriers but the condition is temporary unless there is some predisposing deformity or a chronic focus. These carriers are immune in that they are Schick negative. In the general population true carriers of virulent organisms are less than one in one thousand. Among children about 2 per cent are carriers. Only about 10 per cent of non-contact or non-convalescent carriers show virulent organisms.

4. Pseudo-carriers. In doing extensive carrier work, a number of persons will be found who are carrying organisms which morphologically resemble virulent diphtheria bacilli. Laboratory examinations show that many of these organisms are diphtheroids or non-virulent diphtheria bacilli. In dealing with these individuals it is desirable to determine whether the organisms are virulent and caution should be used in establishing long-term quarantine without doing the "virulent test". On the other hand, carriers of virulent bacilli should be isolated, appropriate treatment instituted. Release cultures should be made before determining the procedure that should be advised.

Symptomatology, Diagnosis, and Complications. - This can be prepared by use of any standard textbook on medicine.

Control of Diphtheria. - Important factors involved in the prevention and control of diphtheria are:

1. Securing swabs and the microscopic examination of cultures from the throats of those who have been exposed to infection.

2. The use of the Schick test.

3. Production of active and passive immunity.

4. The virulence test.

5. Proper use of antitoxin in the treatment of the disease. These elements in the control program are mentioned specifically because of their value and in order to emphasize their use in dealing with diphtheria as a family and community problem.

Is the prophylactic use of diphtheria antitoxin justified? The following statement is made by the Medical Research Council of England on the use of antitoxin in producing passive immunity and is essentially in accord with the attitude of the medical profession of the United States pertaining to this question:

"When the outbreak occurs in a community which is not in close touch with medical observation and when the circumstances are such that the probability of other cases occurring is great, then the use of antitoxin may be considered. Such instances will be confined to communities of children, since adults are so frequently immune naturally that the likelihood of other cases occurring is not so great.

"It may be remarked that the prophylactic injection of antitoxin has certain objections both practical and theoretical. If a child develops

diphtheria after the effect of the antitoxin has passed off, the therapeutic injection may possibly be rendered less effective owing to the presence of precipitins in the blood, and, possibly, may be rendered more toxic by the existence of an acquired hyper-susceptiblity to the horse serum.

"The prophylactic injection of antitoxin should, therefore, be looked upon as a weapon for use in serious emergencies, which are rare, and even then as a palliative which may leave the state of emergency to develop again in three weeks. It will be a matter of individual consideration whether in any particular case it is not more satisfactory to proceed at once along the lines of separation and active immunization of susceptibles by means of Schick's test and toxin-antitoxin mixture rather than to establish a sense of security which is sometimes false."

Since this statement was published the use of toxoid has largely replaced the use of toxin-antitoxin.

Active Immunization.

1. Infants under six months are unsuitable for immunization owing to the frequent presence in their blood of antitoxin passively transmitted by the mother.

2. Children between six months and five years should all be immunized without a Schick test.

3. Children of five years upon entering school, who have not been immunized, and coming under the influence of health officers for the first time should all be immunized without a Schick test, since most of them are susceptible.

4. Older children up to fifteen years should be immunized only if the Schick reaction is positive, since in some schools the number of susceptibles is small and the immunes are likely to suffer more severe non-specific reactions after injection.

5. Children over fifteen years, for the same reason, should be immunized only if the Schick reaction is positive.

If the Schick test, carried out six months after immunization, is still positive, one or two further injections should be given and are usually sufficient to bring about the desired result.

In France in 1923, Ramon introduced a new agent with which he proposed to produce active immunity. The agent he called "anatoxin". English-speaking countries have substituted the word "toxoid" for "anatoxin", which prevents confusion in the use of antitoxin. Toxoid is produced by the detoxification of toxin by adding 4 c.c. of formaldehyde to 1000 c.c. of filtered toxin and heating it from three to six weeks at a temperature of from 30 °to 40° C. This provides a product which is non-toxic and non-sensitizing but which retains its antigenic properties.

Toxoid is given subcutaneously, usually in two or three doses with a two to four weeks' interval. In children under eight years of age the reaction, if any, following the injection of toxoid is slight. However, after the age of eight a marked reaction may occur but without danger. Toxoid is uniformly used in France and Canada, and it is rapidly replacing toxin-antitoxin in the United States and other civilized countries.

Toxoid has the following advantages as compared with toxin-antitoxin:

1. It is about 30 per cent more effective even when only two doses are given.

2. It contains no serum or other animal protein likely to sensitize to a later therapeutic serum injection.

3. It is without local or general reaction in practically all children under seven years of age. Reaction in older children and adults are only unpleasant, not dangerous.

4. It contains no free toxin.

5. It is more stable, retaining its potency for a longer period and is not affected by freezing. The expiration date is usually eighteen months and it may be longer.

6. Immunity occurs much more rapidly (three to six weeks).

7. It is easier to make and costs less.

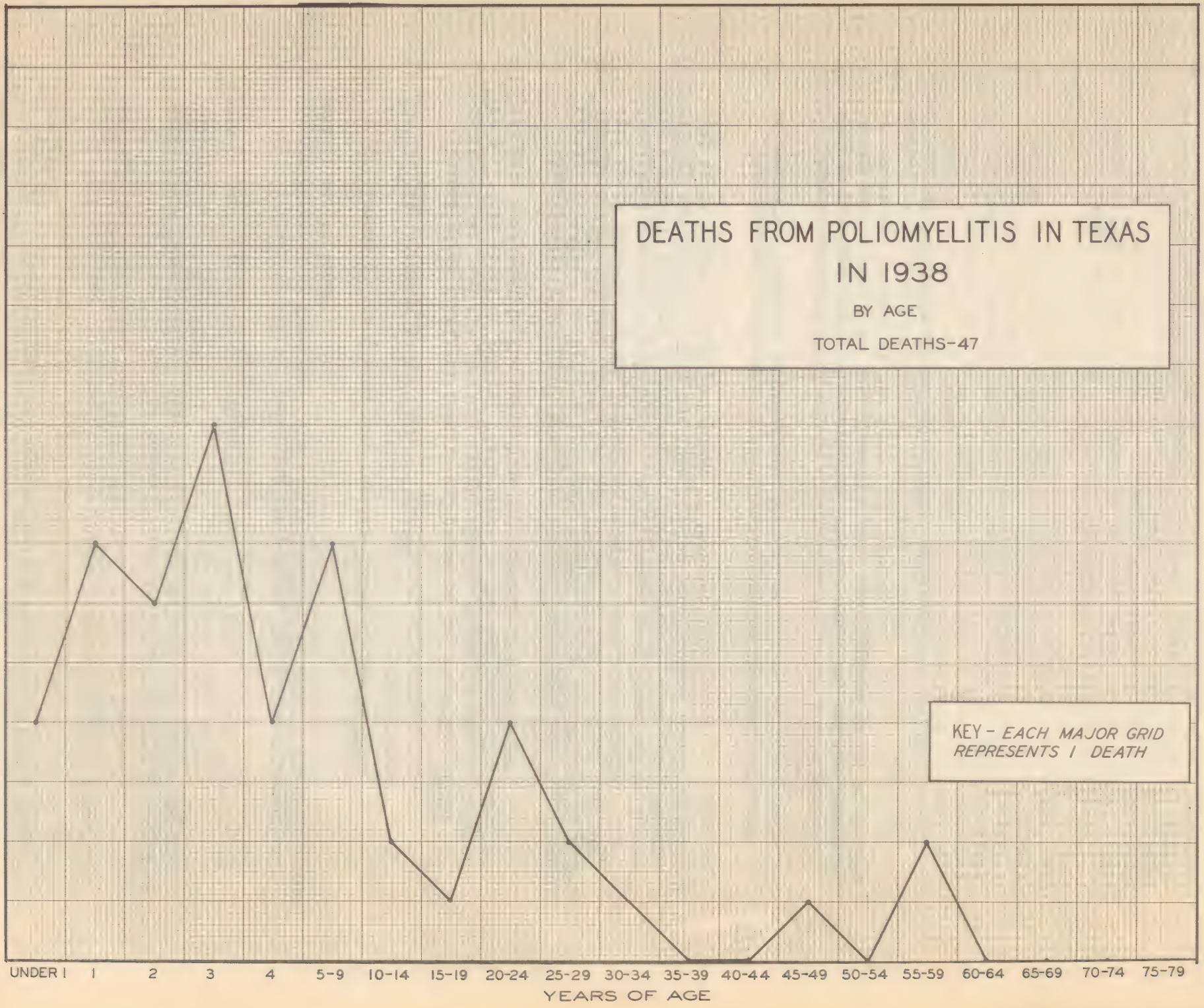
Toxoid gives excellent results and deserves the attention of the health worker, and the physician particularly, since its use is followed by so little local or general disturbance in the age group with which we are most concerned in the prevention and control of diphtheria.

Alum-precipitated toxoid possesses certain advantages and will probably partially replace the soluble or normal toxoid. This is obtained by adding alum potassium sulphate in the process of making toxoid, which combines with or is absorbed by the protein to form an insoluble compound. The insolubility makes it possible to inject larger quantities and as a result the antigenic property is prolonged. The presence of alum slows absorption in the tissues. Only one dose of alum precipitated toxoid is usually used as it causes a high percentage of immunization.

Disinfection. - Concurrent disinfection in the treatment of cases of carriers. Terminal disinfection.

General measures which may be employed in the control of diphtheria, such as personal hygiene, supervision of milk supplies, and the education of the public should be kept in mind. Good oral hygiene is of importance as a means of preventing the spread of infection.

A sore throat is always potentially dangerous.


DEATHS FROM POLIOMYELITIS IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-47
KEY - EACH MAJOR GRID
REPRESENTS 1 DEATH
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

ACUTE ANTERIOR POLIOMYELITIS

Definition - Poliomyelitis is an acute infection, due to a filterable virus, occurring both in epidemic and sporadic form with a special tendency to localization in the anterior horns of the gray matter of the spinal cord; hence the name anterior poliomyelitis. Recent publications present the point of view that it may be a generalized infection, the central nervous system involvement being secondary, but this has not been confirmed.

Etiology - Epidemiological evidence first indicated that poliomyelitis is an infectious disease. Experimental evidence has confirmed this view. It is caused by a living virus. It passes through a filter and produces the disease when injected intracerebrally into monkeys. In 1909 Landsteiner and Popper succeeded in transmitting the disease to monkeys by using the spinal cord of a child who had died of poliomyelitis. Flexner and Lewis in the same year confirmed this result and extended the findings by transmitting the disease through several series of monkeys.

The nature of the virus is unknown. It is present in greatest concentration and can be found in the spinal cord and brain of infected animals and humans. It has been demonstrated in other organs; viz, the mucous membrane of the nose and pharynx, lymph nodes, and is said to have been isolated from the intestine.

The virus is killed by a temperature of 45° to 50° C. in half an hour and by comparatively weak disinfectants. It is not destroyed by low temperatures nor by drying over caustic potash. The virus maintains its virulence in 50 per cent glycerine for long periods.

Geographic Distribution - The area of geographic distribution of poliomyelitis is world-wide. There appears to be a tendency to somewhat higher prevalence of clinically recognizable cases in colder latitudes. The incidence of the disease decreases as the equator is approached, and in the tropics clinical poliomyelitis is a rare disease.

Seasonal Prevalence - Poliomyelitis is usually a warm weather disease. In the United States about 75 per cent of the cases appear within the five months' period from July to November, inclusive, with 50 per cent of the cases falling in the three months of August, September, and October. The curve rises in July and reaches its peak in August or early September and declines rapidly with the advent of cold weather. This is the usual seasonal distribution in endemic areas. Occasionally poliomyelitis will either remain prevalent or appear in the cold months of the year. In the regions south of the equator the seasonal prevalence is the reverse of that for the United States.

Urban and Rural Distribution - According to Frost, one of the most definite characteristics of this disease is its tendency to higher incidence in thinly populated areas; hence, it is more prevalent in rural than urban areas. This is due to the fact that there is less chance for acquired immunity by exposure to repeated small doses of the virus.

Epidemics have been more severe and the case fatality rates have been higher in small towns and rural districts than in the more densely populated cities. Even in cities the disease does not strike especially the crowded districts. In general, the incidence rates are lowest in large cities,

higher in smaller towns, and highest in villages and rural districts.

Age and Sex Distribution - Poliomyelitis is usually considered to be a disease of childhood. On the average about 65 per cent of the cases occur under the age of five years and 95 per cent occur under the age of ten years. The age incidence is likely to be higher in rural than in urban areas.

This disease may occur at any age. During recent epidemics in New York and California the disease tended to attack the older age groups more frequently than had been the case heretofore.

About 56 per cent of the cases occur in males and 44 per cent in females. This ratio has been quite constant at different age levels and in different sections of the country.

Incubation Period - It is difficult to determine the incubation period in the human. Based on the occurrence of multiple cases in families, Aycock estimates that the incubation period is from ten to eighteen days with an average of fourteen days. Epidemiological investigations should consider events taking place about two weeks prior to onset of the case in question.

Modes of Transmission - It is assumed that poliomyelitis is spread by droplet infection from person to person, and the infective agent is discharged from the mouth and nose and enters the body through the same channel. It is considered that the virus is present regularly in the nasopharynx in the first days of illness and disappears as the disease progresses. This would indicate that the period of greatest infectivity is early in the disease.

The prevalence of the infecting agent of poliomyelitis in any community during normal periods is not known, but during epidemics it is likely that a large number of the population harbor the virus for varying periods of time. Only a small number, however, of those individuals invaded by the virus actually develop symptoms of the disease. The temporary carriers are probably more important than the actual case in the spread of the disease. At the present time there is no practical method by which carriers may be detected.

Fairbrother, Hurst and Jungblut have shown that the virus of poliomyelitis travels largely by way of neural pathways; namely, the olfactory nerve endings in the nasal mucous membrane. Once the virus has entered nervous tissue, its migration seems to continue along axonal pathways, passing from the olfactory tract through the hypothalamus, thalamus, and medulla to the spinal cord.

Some workers have presented evidence that the disease may be transmitted through the gastro-intestinal tract. This has been done experimentally under increased intestinal pressure or by direct contact with the mysenteric nerves. This condition is not likely to occur naturally.

Though a few epidemics of poliomyelitis suggestive of milk-borne transmission have been reported by Aycock and others, it is not a common mode of spread of this disease.

Due to the increase in prevalence during the summer months some workers have attempted to connect the transmission of poliomyelitis with an insect vector. It is doubtful whether poliomyelitis is spread by this means.

Direct or indirect contact with previous definite or suspicious cases can be traced only in 20 to 30 per cent of the cases investigated. Recent reports from California and Tennessee indicate that it is possible to obtain evidence of contact in a larger per cent of instances than has heretofore been thought to be possible.

Only exceptionally does poliomyelitis tend to spread within families. In the extensive epidemic in 1916 in New York only 4.3 per cent of 8,634 families attacked had more than one case. When multiple cases occur in a family, they usually come down together or within a short time of each other, signifying simultaneous infection from a common source.

Incidence and Case Fatality Rate - Numerically, poliomyelitis is not a large problem in comparison with other infectious diseases. The sequelae, however, are disabling and crippling in from 25 to 60 per cent of the cases, and for that reason the general public tends to emphasize the problem. According to Leake, the mortality data available for the past three years indicate that poliomyelitis has accounted for about 800 death a year; epidemic meningitis, 1,200; tuberculous meningitis, 2,200; scarlet fever, 2,500; dysentery, from 2,000 to 3,000; diphtheria, 5,000; typhoid fever, 4,300; pertussis, from 4,500 to 7,500; syphilis, 11,000; tuberculosis, 75,000; and automobile accidents, from 28,000 to 36,000.

Based on house to house studies to determine the prevalence of diseases in an entire community, it has been estimated that for the greater part of the United States there are approximately ten paralytic cases per 100,000 population.

The mortality rate in the United States Registration Area varies from that when the incidence of poliomyelitis reaches one per 1,000 population than an epidemic is present. The incidence in any community varies with the immunity in the existing population and the opportunity of spread of the infection. During the epidemic of 1916 in New York the incidence reached 3 per 1,000 population.

Poliomyelitis may be a malignant disease as regards fatality. The case fatality rate will vary markedly from year to year as evidenced by the fact that the rate varied from 30.7 to 5 per cent with an average of 20.8 per cent in 20,568 cases recorded for 38 epidemics which occurred in various parts of the world from 1894 to 1921. In New York City in 1916 there were 8,928 cases and 2,407 deaths, a case fatality rate of 28 per cent. The case fatality rates for Tennessee for small numbers of cases for 1934, 1935, and ten months of 1936 were 19.2 per cent; 45.4 per cent; and 10.5 per cent respectively.

The case fatality rate is likely to be low under the age of five years and increases as age advances.

Immunity - An attack of poliomyelitis usually confers a permanent immunity. It has has been shown that neutralizing antibodies appear in the blood of humans convalescent from poliomyelitis and in monkeys recovered from experimental infection. This can be demonstrated by means of the

neutralization test by which blood serum containing antibodies when mixed with virus renders it non-infective for monkeys. A recent study has shown that only about 60 per cent of the sera examined from that particular group of convalescent individuals contained neutralizing antibodies even though each one had passed through an attack of the disease.

It is thought that a barrier exists between the central nervous and the blood vascular systems and that the virus is usually confined to the nervous system. Harmon and Harkins state that the irregular outflow of the virus from the nervous system during the acute disease accounts for the variable appearance of neutralizing antibodies.

A large percentage of individuals of all ages in the population who have had no obvious exposure to the disease carry neutralizing substances against the virus in the blood stream. The incidence of antibody increases with age. The presence of the substances under these conditions has been interpreted as signifying an immune state and has been produced in the blood as a result of specific contact with subinfective doses of the virus. The relationship between immunity to poliomyelitis and the neutralizing substances is not clear at the present time.

Prevention and Control - The measures that have been applied in the control of poliomyelitis are on the whole unsatisfactory due to lack of definite knowledge of the etiology and mode of transmission. However, certain procedures may be instituted which may be of value.

Notification - All cases should be reported immediately to the health department. This should include the reporting of suspected cases as well as frank cases. Recently the U. S. Public Health Service has requested that cases of this disease be reported as "paralytic" or "non-paralytic".

Isolation of Cases - All cases should be isolated for at least three weeks from date of onset.

Disinfection -Concurrent disinfection of all discharges. The usual procedure should be applied to the disinfection of bedding, utensils, food remnants, etc.

Quarantine - The usual procedure is to quarantine all children in the household for a period of three weeks. Under certain circumstances adults in contact with cases may also be quarantined.

Prevention of the introduction of the disease from the outside in the case of children's institutions by quarantine may be advisable.

Voluntary "quarantine" of young children by parents may be advocated.

Educational measures should be employed in an attempt to give the public accurate information concerning the disease and to keep the public informed as to the progress of the epidemic. An attempt should be made to prevent the usual fear which is immediately aroused upon the appearance of the disease in the community. Cooperation should be encouraged in avoiding any unnecessary exposure of children in crowds, in travel and in visiting. Advice should be given as to keeping children well by avoiding undue or unnecessary fatigue, trauma and gastro-intestinal disturbances. Nothing is to be gained, as a rule, by closing schools.

Diagnostic Facilities - Health department should be prepared to furnish diagnostic service and facilities through personnel thoroughly familiar with poliomyelitis. During an epidemic it is only by this means that early cases can be detected and the disease differentiated from numerous other acute conditions which may resemble poliomyelitis and be confused with it in the early stages.

Vaccination - For the past two decades attempts have been made to produce an active immunizing agent against poliomyelitis by means of attenuating the virus by means of chemicals, heat, desiccation and absorption to alumina gel. Following the use of these agents, neutralizing antibodies could be demonstrated in experimental animals. Following inoculation with the agents, some of the monkeys developed poliomyelitis after the intranasal or intracerebral application of virulent virus as a test of immunity.

In view of the fact that the virus has difficulty in getting from the blood stream to the central nervous system, future experiments should explore the possibility of using the immunizing agent so that it will get through the blood vascular system and come in contact with the central nervous system. There appears to be an undetermined mechanism which prevents this process.

Sprays - The normal mucous membrane and its secretions represent protective barriers against the invasion of pathogenic organisms. Any chemical substance or manipulation which may cause irritation or trauma are likely to assist the entrance of infective agents. However, Armstrong and his associates recently have shown experimentally that a spray solution of picric acid and sodium aluminum sulphate would protect monkeys against the virus of poliomyelitis instilled into the nasal cavities, the solution forming a protective coating over the mucous membrane. The possibility of creating an artificial blockade by this means to prevent the entrance of the virus into the body is still an experimental procedure, but it is available for use by physicians at their discretion. No spray should be used unless advised by the physician.

After Care and Rehabilitation - While it is not the duty of health officials to treat cases of poliomyelitis, they should be acquainted with the scientific procedures and be able to render any assistance and give advice in order that disability may be reduced to a minimum. Certain points such as placing the paralyzed extremities at rest in a normal position to prevent contractures, and the prevention of any undue manipulation or unnecessary treatment until the acute stage of the disease has subsided should be emphasized. The problem resolves itself into one for the orthopedic specialist and the public should be informed of the great benefit which may accrue if such a specialist is called in to supervise the case before contractures and atrophy occur. Probably an orthopedist should see each paralytic case of the disease as soon as the diagnosis is made.

Programs for the rehabilitation of crippled children are being conducted by many private organizations and by governmental agencies in a large part of the United States. If it became necessary to provide follow-up for a considerable number of poliomyelitis cases, the public should be informed about such services and the cases should be followed up so that these agencies can work efficiently. A nurse with special training in physiotherapy should be made available to the local health service for this purpose.

TYPHOID FEVER

Typhoid fever is an acute, specific, generalized infection, usually characterized by a gradual increase in temperature, with or without diarrhea, rose-colored eruption, slow pulse, and tenderness over the abdomen.

The incubation period varies from seven to twenty-eight days; average ten to fourteen days. This bears an important relationship to the epidemiological studies. The average duration of the fever is about twenty-five days; case fatality is about ten per cent.

Let us review just briefly some of the morphological and biological characters of the causative organism, B. typhosus, or Eberthella typhi as it is now known in recognition of its description in 1880 by Eberth. It is a Gram negative, non-spore forming, motile rod. It is an aerobic, facultative anaerobe; does not produce indol, and fails to ferment either lactose or saccharose. These latter factors are of importance in differentiating this organism from certain others of the enteric group. This organism is not difficult to cultivate on simple laboratory media, but does exhibit frequent morphological variations. The significance of these forms is not clear; there is no evidence that they are involution forms; it is probable that they represent phases in the life cycle. The type of variability with which we are concerned is the dissociative changes from "smooth" to "rough", and which we shall develop further in this paper.

Typhoid bacilli does not thrive well outside the human body, but with favorable temperature, moisture, and food supply will live sufficiently long for indirect transmission, especially in water, milk, and certain other food supplies.

The portal of entry is the mouth; excretion is through the feces and urine. The organism has been recovered from the sputum, and certain other body fluids. The gall-bladder, biliary passages, marrow, spleen, and mesenteric lymph nodes are the principal reservoirs of the human body.

Typhoid fever is transmitted by direct and indirect contact. It may be spread directly from a person who has or has had the disease to one who is susceptible to it. It may be spread by indirect contact, in water, milk, milk products, oysters, vegetables, and other foods; also, by flies and fingers contaminated by human excreta.

The mode of spread may be referred to as:

1. Water borne.
2. Milk borne.
3. Contact infections:
    a. Cases - acute, atypical, mild, or unrecognized.
    b. Carriers.
    c. Objects contaminated - utensils.

4. Other foods contaminated.

5. Fly borne.

Water borne:

There is a seasonal variation. It may occur in the fall, winter, or spring, but usually in the late winter or the early spring. The outbreak is usually explosive, with gradual increase of incidence until the peak is reached, and a gradual decline if the source of contamination has been removed. There is a general distribution of cases throughout the area supplied by the polluted water - may be occasional outside cases. There is relatively a larger per cent of the cases among adults. Epidemics from this source are almost always preceded by premonitory diarrhea. It is usually possible to indict this source by a preliminary survey. The evidence may be confirmed by:

A careful study of area with reference to source of contamination.

A bacteriological examination of the water may show colon bacilli.

A chemical analysis showing nitrites also indicates pollution.

In suspected pollution of water, immediate action should be taken to insure boiling of water for domestic purposes.

Milk borne:

Sources of the infection include:

1. Delivery of milk to premises where a case exists.

2. A carrier employed on the dairy or farm producing milk sold in the community, raw or imperfectly pasteurized.

3. Washing utensils with polluted water; cleansing udders with contaminated water.

4. Milk may be contaminated by flies, or by dirt where there is improper protection.

5. May be defective pasteurization.

6. May be contaminated by the consumer.

Milk is a favorable medium for the rapid multiplication of the organisms; therefore, massive doses may be ingested. Immunity may break down, even in recently vaccinated individuals.

Cases usually will be found along the route of a certain distributor. There is no seasonal variation as seen in the water-borne. The incubation period usually is shorter because of large doses of the organisms. A larger per cent of the cases occur among women and children. Often more than one case in a family occurs about the same time. More cases usually occur in families consuming considerable milk. The outbreak is usually sudden, with a rapid rise to the peak, and a corresponding decline.

A thorough survey of the personnel, methods of distribution, and consumption should be made in order to determine the source.

Contact infection:

This may be the result of association with an acute case of the disease, a mild or unrecognized case, or with a carrier. The carrier problem in the transmission and control of typhoid fever has become one of great importance. From two to four per cent of persons who have had the disease continue to discharge the organisms in the excreta. Every case, therefore, is a potential carrier.

Outbreak due to carrier:

Usually, isolated or sporadic cases occur. The number of cases is usually small, and intervals between cases may be long. There is no seasonal variation. The outbreak due to a carrier is usually in an institution, or camp, where the carrier has to do with preparation or handling of food.

Fly-borne:

The characteristics are similar to the carrier outbreak, except that fly transmission occurs more in the warm months of the year. This source is usually arrived at by elimination of other possibilities, as water and milk, plus insanitary conditions in the environment.

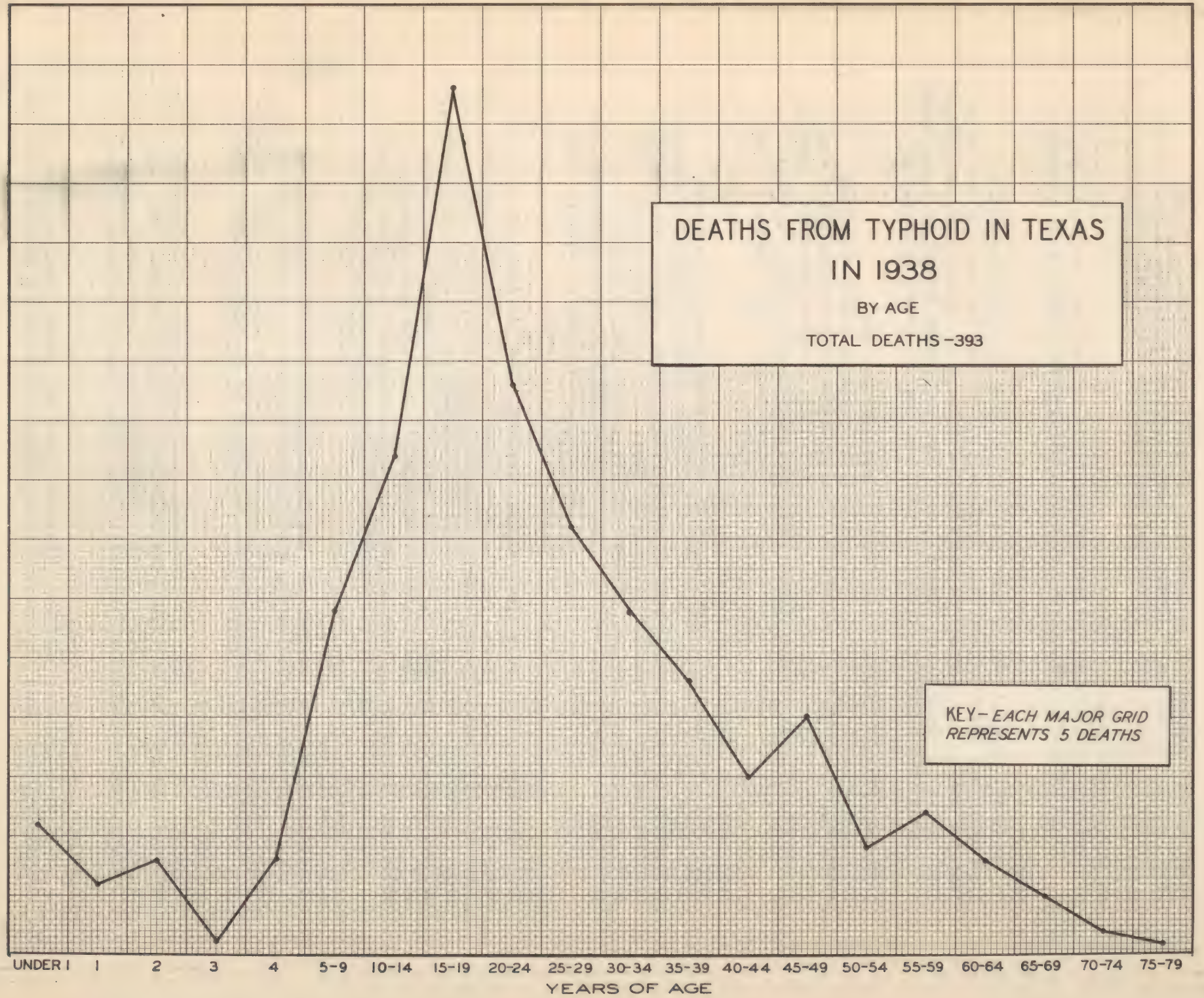
In a review of the methods and mechanisms of artificial immunization against typhoid fever and its relation to the lowered incidence of this disease, it is well that we do not lose sight of many other factors which have abundantly contributed. In fact, it is considered by many that improvement of water supplies, sewage disposal, and education of the public resulting in general knowledge of the transmission avenues, have been as important as vaccination.

It is proper to review briefly the history of typhoid vaccination, and to discuss certain factors which may influence the protective action of vaccines, that is, their efficiency as a prophylactic agent, and some problems in the serological diagnosis which have arisen as a result of these vaccinations.

The use of vaccines in the prevention of certain other diseases naturally suggested the use of such agents in typhoid.

As early as 1888, Widal, and others, showed that animals could be protected against lethal doses of virulent typhoid bacilli by previous inoculation with killed typhoid organisms, allowing time, of course, for this immunity to be established. Next, it was found that the serum of these vaccinated animals contained specific anti-substances which were identical with those demonstrable in the serum of persons recently recovered from typhoid fever. Out of these observations was developed the possibility of human immunization.

In 1896, Wright, in England, began to use killed typhoid suspensions in human inoculations. He observed that the procedure was safe, and was followed by demonstrable antibodies in the blood of vaccinated individuals.


DEATHS FROM TYPHOID IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS - 393
KEY - EACH MAJOR GRID REPRESENTS 5 DEATHS
UNDER 1
1
2
3
4
5-9
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75-79
YEARS OF AGE

The following year this same worker suggested the use of this vaccine in the armies of Great Britian. This was done, but these first efforts did not appear to have any appreciable effect on the incidence of typhoid.

Following this apparent failure in mass immunization, work was begun on better methods of preparation and standardization of vaccines. More suitable strains were selected, and methods of cultivation were improved. It is not improbable that the phenomenon of dissociation was responsible for the early failures.

In 1908, typhoid vaccination was introduced in the Army of the United States, using the English "Rawlins" strain of typhoid bacilli. This strain had been selected for use in the British army because it produced in man a satisfactory titer of antibodies, and possessed what we now designate as smooth qualities.

At first the Army vaccine consisted of a saline suspension of the organisms, killed at 53 degrees Centigrade for one hour, and standardized at 1000 million organisms per c.c. Actually, there were only about 500 million per c.c. In 1917, 750 million each of para-typhoid A and B were added. Reactions with this vaccine were considerable. In 1927, para-typhoid B was dropped, and typhoid increased accurately to 750 million. It was now used in this state, giving three doses, each at weekly intervals. It is the practice at this time to give one-half c.c., followed by one c.c., doses at weekly intervals.

In 1913, the Army had, in 80,000 men, only four cases of typhoid fever. From 1909 to 1914, there was only one death in the Army from typhoid fever, while the civilian rate for the same period was 16.5 per 100,000; a substantial reduction, however, from 35 in 1900. In 1900, typhoid fever was the fourth on the list as cause of death in the United States, but in 1935, had reached twelfth place. During the World War (April, 1917, to November, 1918) in 4,122,930 men in the Armies of the United States, there were 1529 cases of typhoid fever with 122 deaths.

One attack of typhoid fever usually confers a life-time immunity. It is estimated that a second attack may occur in 2 to 3 per cent of cases. As we evaluate the immunity as produced by actual disease experience, and by vaccination, let us not lose sight of the fact that these individuals are from time to time subjected to sub-infective doses of organisms which doubtless aids in fortification of the immune mechanisms.

Circulating antibodies usually disappear from the serum of recovered individuals in about seven months, and in the vaccinated person in a few weeks. Immunity cannot, therefore, be explained on a basis of circulating antibodies. Then where are these substances? They must be in association with the cells of the body; rather, let us say, the cells of the reticulo-endothelial system.

It is generally agreed that we cannot explain all immunity on a basis of antibodies. The cellular mechanism also plays an important part, to which we shall refer later.

It is not our purpose to outline the technical features of the Widal test, but it is desired to point out some problems which have arisen in the serological diagnosis in the sometime vaccinated individual. It was

difficult to determine whether antisubstances demonstrable in the serum were due to infection with the organism, or to the effect of the vaccine, or previous experience with the disease. The problem is further complicated by what is termed "anamnestic reactions". These latter reactions are due to a return, or "recall", to the circulating blood of antibodies produced in response to a previously introduced antigen under the stimulus of a subsequent antigen. This second stimulus may not be the antigen which produced the existing antibodies. For example, an individual contracts Malta fever; he was vaccinated against typhoid fever six months ago; a Widal might now show a titer of agglutination which we would be inclined to accept as diagnostic.

At this time let us mention some of the work which has been done in an effort to differentiate these agglutinations. We refer here to work with the H, or flagellar antigen, and the O, or somatic antigen, and the Vi antibody.

It was observed by Felix, in 1924, that vaccination produced almost exclusively the "H" type of agglutinins, while the infection produced chiefly the "O" type of agglutinins, and that we might differentiate these reactions by the use of the homologous antigen, observing the type of flocculation produced.

We have already noted the fact of morphological variation in typhoid bacilli grown on artificial media. Arkwright has shown that the chief variants are:

Smooth motile; smooth non-motile
Rough motile; rough non-motile

Antigenic changes, changes in pathogenicity and bio-chemical activities are results of these dissociations.

Further studies by Felix have shown that there are smooth "O" strains which are inagglutinable, and other which are agglutinable in "O" antiserum, also, that only these inagglutinable virulent organisms produced sera which would agglutinate and protect. The inference is that the virulent organisms produce a third antigenic factor which he calls Vi. When the sera of animals immunized with virulent strains are prepared, or, as we would say, set up, with agglutinable and inagglutinable cultures, the agglutinable strains take out both "H" and "O" antibodies, but not the Vi; on the other hand, Vi strains take out the Vi antibody, but very little of the "H" and "O". The Vi antigen is present only in the living, non-heated organisms, is destroyed by 60 degrees Centigrade, and is only partially preserved by formalin. The conclusion is that vaccines should be prepared from Vi antigen, since it is this fraction which is responsible for the production of protective antisubstances, and gives us a more accurate differentiation of the vaccinated individual.

Siler has worked with many of the strains which have been used in the preparation of vaccines at the Army Medical School. In protection tests he has found that vaccines prepared from avirulent strains have failed to protect mice from infection when inoculated with virulent organisms. He has found, also, that virulent strains differ in their antigenic properties. He concludes that immunogenic property in typhoid bacilli is closely associated with virulence, and that heat killed, tricresol vaccines give better protection.

Hawley and Simmons have studied the effectiveness of the Army and Navy vaccines in relation to dosage. They believe that the "Rawlins" strain is as efficient today as it ever was, and that decrease in efficiency can be explained on a basis of dosage.

It is a well-established fact that there are many antigenic components in a bacterial cell, the principal ones being the protein and polysaccharide fractions; within these fractions there appears to be a multiplicity of sub-fractions, as indicated by Furth and Landsteiner working with the typhoid bacillus.

The studies up to this time have suggested that among these various fractions of the bacterial cell, there is a specific, or essential, immunizing antigen.

Felton and Wakeman have made an investigation of the active immunity producing properties, and some of the chemical characteristics of the fractionated typhoid bacillus. They have found that the killing temperatures and chemical preservation may alter the antigenic properties of a vaccine; that certain antigenically inert substances, as lipoids, may actually inhibit the immunizing mechanism, and that certain growth products of the organism in culture may do the same thing. In their preliminary report, it is indicated that an acetone extracted polysaccharide fraction is the essential immunizing agent.

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OUTLINE FOR THE CONTROL OF TYPHOID FEVER

I. MEASURES OF IMMEDIATE IMPORTANCE

A. Records

1. Report cards in the hands of physicians.
2. Epidemiological investigation.
   a. Spot maps.
   b. Classification of findings.
3. Tabulation of cases by days, weeks, months.
4. Tabulation of cases by age, sex, color.
5. Tabulation of deaths by age, sex, color.

B. Notification

1. Compulsory reporting of cases.

C. Case findings on unreported cases.

D. Investigation

1. Investigation of cases as soon as possible.
2. Complete epidemiological history on every case.
3. Confirmation of diagnoses.

E. Management of cases

1. Isolation, screening.
2. Hospitalization.

3. Concurrent disinfection:
   a. Stools and urine.
   b. Bed linen and clothing.
   c. Utensils.
   d. Sputum and purulent discharge.
   e. Container with disinfecting solution for use of physician and attendant.
   f. No containers to be removed by milkmen.
   g. Frequent calls upon case.
   h. Terminal disinfection.
   i. Release cultures.

F. Control of contacts.
   1. Vaccination regardless of history of typhoid.
   2. Examination of stools and urine if indicated.
   3. No member allowed to handle food until determined safe to do so, especially in food establishments.

G. Control of carriers.

   1. Determination and registration of carriers.
   2. Check on known carriers twice yearly.
   3. Instruct carriers in hygiene.
   4. Prohibition of food handling by carriers.
   5. Efforts to eliminate carrier condition.

II. MEASURES OF GENERAL IMPORTANCE

A. Sanitation.

   1. Excreta disposal.
      a. Number of families connected sewer, privies, etc.
      b. Extension of sewer connections.
      c. Compulsory connection with sewerage system.
      d. Regulation of privy construction.
      e. Inspection.
      f. Treatment of sewage.

   2. Water supply.
      a. Frequent laboratory examination of water.
      b. Frequent inspection of plant and system.
      c. Filtration and chlorination.
      d. Swimming pools: inspection, examination, chlorination.

   3. Milk and dairy products.
      a. Inspection of dairies and examination of milk handlers.
      b. Bacteriological examination of milk.
      c. Pasteurization.

   4. Other foods: Shellfish and vegetables.

   5. Foods in general.
      a. Examination of food handlers.
      b. Inspection of food establishments.

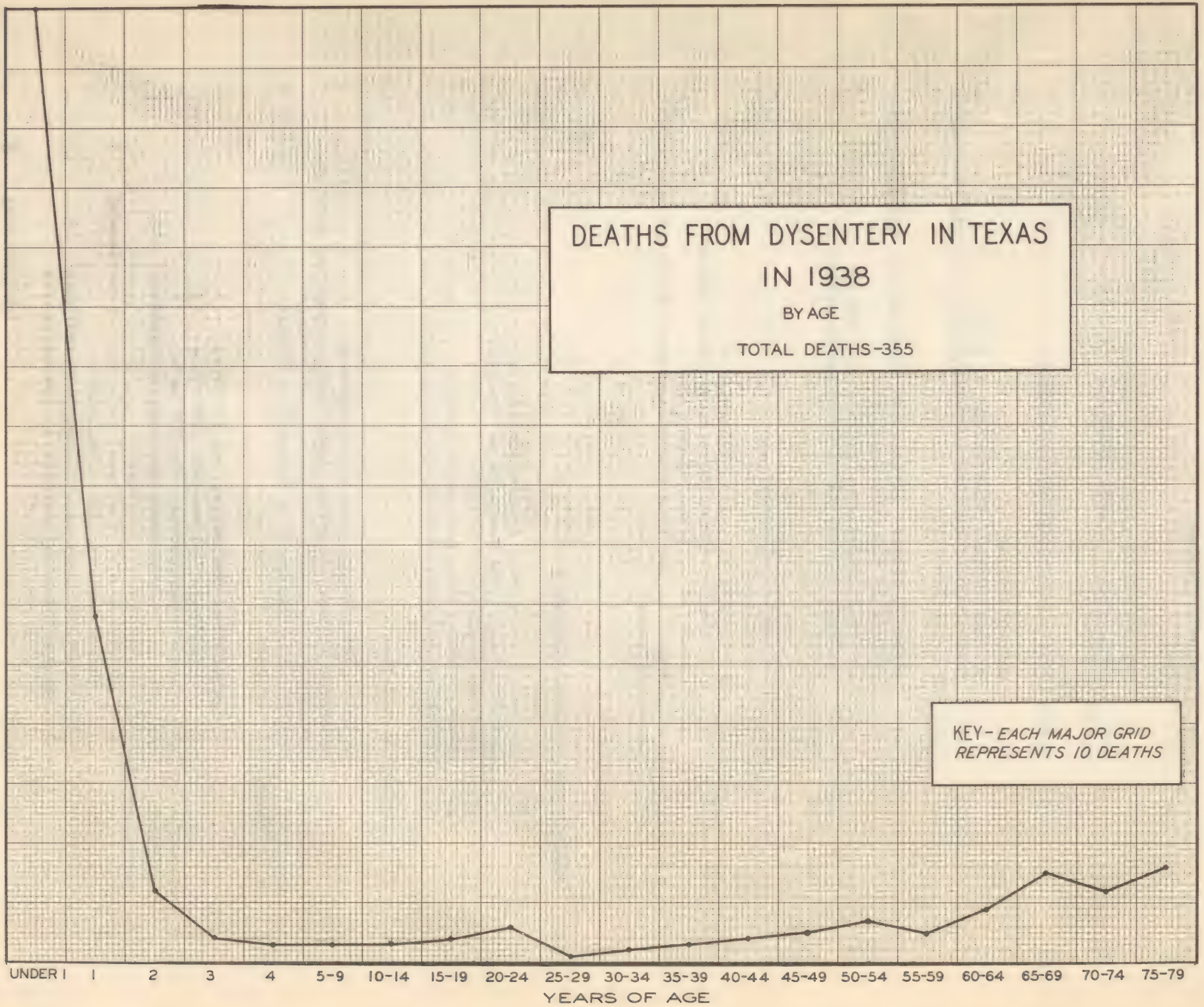
   6. Fly control.
      a. Garbage disposal.
      b. Screening.

B. Vaccination.

1. Establishment of clinics for vaccination.
2. Vaccination of school children.

C. Education.

1. Responsibilities and dangers.
   a. Cause and how transmitted.
   b. Principal sources of infection.
   c. Value of sanitation and vaccination.


DEATHS FROM DYSENTERY IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-355
KEY- EACH MAJOR GRID REPRESENTS 10 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

BACILLARY AND AMOEBIC DYSENTERY

Dysentery is a symptom complex consisting of diarrhea with blood and mucous in the stools, usually associated with abdominal pain and tenesmus. The lesions are usually limited to the colon, appear as areas of ulceration, discreet in the amoebic type, with normal mucosa surrounding. In the bacillary variety the mucosa between lesions tends to be hyperemic.

Since ancient times dysentery has occurred sporadically, and in epidemics. The epidemics have usually been associated with hot climates, filth, and general unsanitary conditions in the environment. In 1892, Councilman differentiated the pathology of the amoebic and bacillary dysenteries, and in 1898, Shiga proved the specific bacillus now known as the cause of the Shiga type of dysentery.

With differentiation of the amoebic and bacillary types of dysentery, our knowledge of the transmission, and methods of control, much has been done to lower the incidence of these diseases. Epidemics still occur both in the tropics and in the temperate regions where proper sanitation is not observed.

The dysentery bacilli belong to the large group of intestinal Gram negative rods, differing from the typhoid and colon group in that they are non-motile, and possess certain cultural and serological differences.

There are two main groups of dysentery bacilli. One group includes only the Shiga bacillus, is more virulent, possesses a neurotoxin as well as an enterotixin. The other group includes the Flexner, Hiss-Russell (Y), Sonne and other strains, all of which are less virulent than the Shiga, possess only an enterotoxin and ferment mannite in culture. Some mildly pathogenic strains usually produce only a mucous diarrhea without blood. The bacilli in man multiply in the small intestine. The toxins are absorbed, and act on special tissues. The neurotoxin produces paralysis in animals and stupor in man. The enterotoxin acts mainly upon the mucosa of the colon during its excretion from the blood, and causes a diphtheritic inflammation. In animals this can be produced by intravenous injection of the toxin, but not by ingestion of cultures. In man the bacilli are found in the dysenteric stools, sometimes almost in pure culture in acute cases, and may persist in chronic ulcers producing the carrier state. Outside the body the organisms are easily destroyed, but may survive in water, milk, or moist soil three weeks if not exposed to sun. They will live two to five days in the intestines of flies.

Incubation Period. -

Two to seven days.

Period of communicability. -

Usually until diarrhea ceases. Actually until no more organisms are excreted in the feces.

Incidence. -

All ages are susceptible, and no racial immunity is known. An exact estimate of incidence is difficult to obtain because many mild cases are not seen by physicians. High atmospheric temperatures favor spread of the organisms because of their survival outside the body and the presence of flies. Therefore, epidemics and sporadic cases are most common in summer and in tropical countries. Undoubtedly scores of small epidemics occur in this country

every year, and many thousands of people suffer from the disease. The majority of cases of summer diarrhea in infants is caused by dysentery bacilli.

Mortality. -

Severity of attacks depends mainly upon the variety of organism involved. The very young and the very old show the highest case fatality rates. In Shiga epidemics the mortality is often 30 per cent or more. In epidemics with the mannite-fermenting group it is usually not over 5 per cent.

Immunity. -

Agglutinins appear in the blood during convalescence and remain for one to two years. There is probably an immunity of uncertain duration against the strain of organism causing the disease, but not against other strains. During epidemics some people seem to develop agglutinins without a clinical attack.

Sources and Modes of Infection. -

Man becomes infected from water or milk, from food contaminated by flies or human carriers, or by direct contact with the discharges from active cases. Direct contact and flies are more important than in typhoid fever because of the enormous numbers of organisms present in the dysenteric stools. Monkeys are the only lower animals known to harbor the organisms. They sometimes suffer from acute bacillary dysentery.

Carriers. -

Ordinarily the bacilli disappear from the stools within a week or two after cessation of symptoms, but about 3 per cent of cases become chronic, with localized ulcers which may harbor the bacilli for years. Shiga cases are more likely to become chronic. Relapses may occur. The chronic relapsing case most often becomes a carrier. Chronic carriers may have no symptoms, and may excrete bacilli at irregular intervals. They probably have small lesions in the colon. Two to five per cent of cases become carriers after an epidemic. Chronic cases and carriers are responsible for the persistence of the disease from year to year. They are dangerous as food-handlers and especially if proper excreta disposal in not observed. Healthy carriers without a history of dysentery are rare. Some cases of chronic ulcerative colitis are really chronic bacillary dysentery.

Epidemiology. -

In the middle ages there are said to have been very extensive epidemics, but in recent times they have been confined to smaller groups. In wars dysentery has been more dangerous than bullets. There were over 1,500,000 cases in the federal army during the Civil War and 57,000 deaths. The longer troops are together the higher the morbidity rate and case fatality rate. There were serious epidemics during the World War. Prisons and asylums frequently have epidemics. Towns in unsanitated regions have epidemics, and contaminated city water supplies have given rise to them. Every year many epidemics occur in rural areas and villages in the Southern States. In the tropics epidemics usually coincide with the rainy season. The maximum incidence coincides with that of the housefly.

Prevention. -

(1) In hospitals, institutions and camps great care must be taken to avoid spread of infection from active cases.

(2) Attendants on cases should observe strict precautions.

(3) Fecal discharges of patients should be disinfected.

(4) Patients should not be discharged until stool culture is negative for dysentery bacilli on three successive examinations not less than 24 hours apart.

(5) Chronic cases and carriers in camps and institutions should be isolated until free from dysentery bacilli.

(6) Food-handlers in camps and institutions should be examined before employment.

(7) All cases of diarrhea in camps, institutions, asylums or in communities where sanitation is poor should be looked upon with suspicion.

(8) Vaccination has a definite value in checking an actual outbreak of the disease and in reducing mortality but only lasts from two to three months. It has not been used extensively because other measures are usually sufficient and because of severe reactions following vaccination. Recently an "anavaccine" from which the toxicity has been removed has been reported to give good results.

(9) Prevention of fly breeding and exclusion of flies from habitations and food, and especially from the dysenteric discharges of acute cases.

(10) Early efficient treatment to avoid the carrier state.

Amoebic Dysentery

Endamoeba histolytica is the only pathogenic amoeba. It exists in two forms, trophozoite or motile form and cyst. The trophozoite measures 10 to 30 micra in diameter, has hyaline pseudopods, moves rapidly at body temperature, and often contains red blood cells when found in a dysenteric stool. It soon dies after leaving the body. The cyst measures 6 to 15 micra in diameter, when mature has four nuclei of characteristic structure and is resistant to low temperature if kept in a moist condition. It is quickly killed by drying. It will survive two days in the intestines of a fly, and a month or more in clean water. Infection takes place only by the ingestion of cysts. Reaching the cecum these excyst and under proper conditions, which are poorly understood, invade the intestinal wall and produce ulcers. Symptoms are dependent upon the extent of ulceration, which may occur anywhere in the large intestine and rarely in the ileum. It is probable that different strains of E. histolytica vary greatly in their invasive power and therefore in the frequency with which they produce symptoms.

Incidence.

Amoebiasis, that is, the presence of E. histolytica in the stools, is widespread throughout the world. About 7 to 10 per cent of people are infected in temperate zones, 20 to 80 per cent in the tropics. The incidence is 40 to 50 per cent in some isolated communities in the Southern States. In unsanitated areas the incidence of amoebic infection is rapidly built up during childhood and is maintained at a fairly constant level throughout the adult decades. Acute amoebic dysentery is commonest in young and middle-aged adults but may occur at any age. The sexes are equally affected. All races are susceptible, but persons going to an area where much amoebic dysentery occurs seem to be especially susceptible. Most of those harboring the parasite are apparently healthy, even though they are infected with a strain of amoeba which is highly pathogenic. Acute amoebic dysentery may occur anywhere but is much more common in the tropics than in temperate zones and in the summer than in the winter. The factors responsible for this are not known, but they are probably related to the condition of the host, to food or to bacterial infections of the intestinal tract. Secondary bacterial infection may be important in producing extensive ulceration.

Incubation Period.

Seven to one hundred days, and some infected persons never develop clinical symptoms.

Period of Communicability.

As long as cysts are excreted in the stools, which may be a lifetime.

Sources and Modes of Infection.

Infected food-handlers, polluted water supplies, infected feces exposed to flies, or human fecal material used as fertilizer in gardens. Food-handlers and flies are common sources of infection; polluted water may be a source of infection from wells, springs, or streams, or from cross connections between sewers and pure water supplies in cities. Milk is rarely if ever a source of infection. The slow multiplication of the

organisms, their relatively small number, the slow development of the disease and the absence of spread from one acute case to another make amoebic dysentery more sporadic and less epidemic than bacillary dysentery. Reservoir animal hosts of E. histolytica are a possible source of human infection but this is undoubtedly rare. Monkeys, dogs, cats, and rats have been suffering from acute amoebic dysentery. Hogs sometimes harbor E. histolytica without symptoms. It is not known whether these strains would produce lesions in man.

Epidemiology.

Epidemics of acute amoebic dysentery of small and large proportions have been reported. The source of infection and the mode of transmission determine the epidemic picture which is produced. In India a multitude of polluted wells causes a highly endemic condition in which acute cases occur constantly. In the Philippines soldiers on the march drank water from polluted streams, producing an epidemic. On the Mexican border an epidemic among troops seemed to have been produced by flies carrying infected fecal material from insanitary privies to food. The same condition seemed to cause a slow spread of the infection through a rural community in Tennessee. In Chicago pollution of constantly recirculated water in a hotel by a sewer connection produced an epidemic in which the transient guests scattered to all parts of the country before their clinical symptoms developed. A food-handler may infect:

(1) a whole family

(2) many persons in an institution, or

(3) occasional guests at a hotel.

The long incubation period and the irregularity of symptoms often clouds the epidemic picture and hides the source of infection.

Immunology.

Complement-fixing antibodies are present in most infected persons. An acute infection is usually followed by a latent period of partial cure, which suggests local or partial or temporary immunity, but the frequency of recurrences suggests that a general immunity is not developed. Monkeys have been reinfected after cure. Spontaneous permanent disappearance of the infection without treatment has not been reported in man, but has occurred in certain animals which are not very suitable hosts, such as dogs and rats; and, with certain mild strains of E. histolytica has occurred in kittens which regularly die from highly pathogenic strains.

Prevention.

The acute case does not usually pass cysts but may do so. Although he is not usually a source of infection, his excreta and bed linen should be sterilized and attendants should observe proper precautions. The cyst carrier is the only source of transmission. It would be ideal if all public food-handlers could be examined to detect carriers, but under ordinary conditions this is not feasible. Frequent skillful examination would be necessary to make it effective. All detected carriers should be treated regardless of whether they have ever had symptoms. Cases and carriers should be warned that one course of treatment usually does not cure and should be followed at intervals for one year to be sure of a cure.

Vegetables should not be eaten raw in regions where human excreta are used for fertilizer. Sanitary disposal of feces and their protection from flies is most important. Fly-breeding should be eliminated and flies excluded from houses and food. Private and public water supplies should be protected from pollution, and city health authorities should see to it that cross-connections are not allowed to exist between sewer pipes and pure water supplies.

Diagnosis of Type of Dysentery

Clinical Course.

Bacillary dysentery is usually more acute than amoebic, with higher temperature, more prostration, and toxemia, and shorter in duration.

Gross Appearance of Stools.

Bacillary stools have a pink, purulent appearance, blood and pus intimately mixed, and mucus friable. Amoebic stools are more watery, with relatively little pus; blood and mucus are not so intimately mixed and mucus is more tenacious.

Microscopic Appearance of Stools.

Bacillary stools contain many polymorphonuclear leucocytes and many large mononuclears, both types usually showing degeneration. Amoebic stools have fewer polymorphonuclears, red blood cells are more in masses, and motile amoebae are usually present. Neither the gross nor the microscopic appearance, however, is absolutely reliable if amoebae are not found.

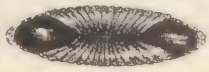
Stool Cultures.

In the absence of amoebae, cultures should always be made. Amoebae may appear on culture in suitable media when not found microscopically. Culture should be made for dysentery bacilli. Special technique is required to isolate either amoebae or dysentery bacilli, and directions should be followed carefully. Suitable fresh specimens, proper treatment of material, and proper media are essential. Routine cultures for dysentery bacilli often fail for lack of these precautions.

In chronic dysentery proctoscopic examinations and examination of scrapings from ulcers both microscopically and culturally are often important. Sometimes repeated examinations are necessary. Some cases show both amoebae and dysentery bacilli.

LIFE STAGES AND DEVELOPMENT OF THE MOSQUITO

ANOPHELES
MALARIA-CARRYING MOSQUITO

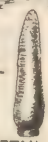


DETAIL OF EGG.

GROUP OF ANOPHELES EGGS.

CULEX
PEST OR COMMON MOSQUITO

EGG RAFT.



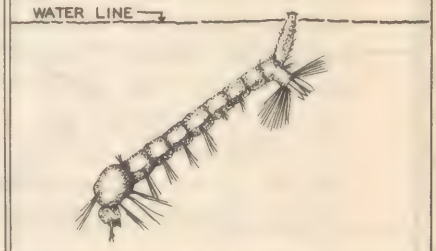
DETAIL OF EGG.

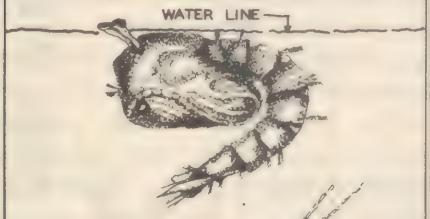
EGG

A FEMALE MOSQUITO AFTER SUCKING A BLOOD MEAL, LAYS HER EGGS ON WATER SURFACE.
←— ANOPHELES MOSQUITO EGGS ARE NOT HELD TOGETHER BUT EACH HAS A SEPARATE FLOAT.
CULEX —→
OR COMMON MOSQUITO EGGS ARE HELD TOGETHER IN RAFTS.
TEMPERATURE OF WATER AND SEASON OF YEAR INFLUENCE THE HATCHING TIME—USUALLY 6 HOURS TO 3 DAYS.

LARVAE

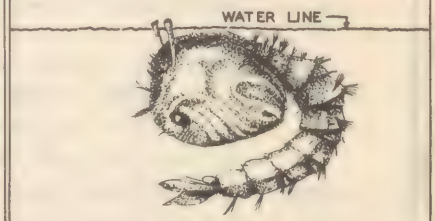
MOSQUITO LARVAE OR WIGGLE-TAILS ARE HATCHED FROM MOSQUITO EGGS.
LARVAE ARE CLASSIFIED AS TO SIZE AS FOLLOWS: 1. THOSE JUST HATCHED; 2. ONE QUARTER SIZE; 3. ONE HALF SIZE; 4. THREE QUARTER SIZE; 5. FULL GROWN LARVAE.
←— ANOPHELES LARVAE LIE PARALLEL TO WATER SURFACE.
CULEX —→
OR COMMON MOSQUITO LARVAE LIE AT AN ANGLE TO WATER SURFACE.
TEMPERATURE OF WATER, SEASON OF YEAR, AND THE FOOD SUPPLY GOVERN LARVAE GROWTH TIME WHICH VARIES FROM 7 TO 90 DAYS.





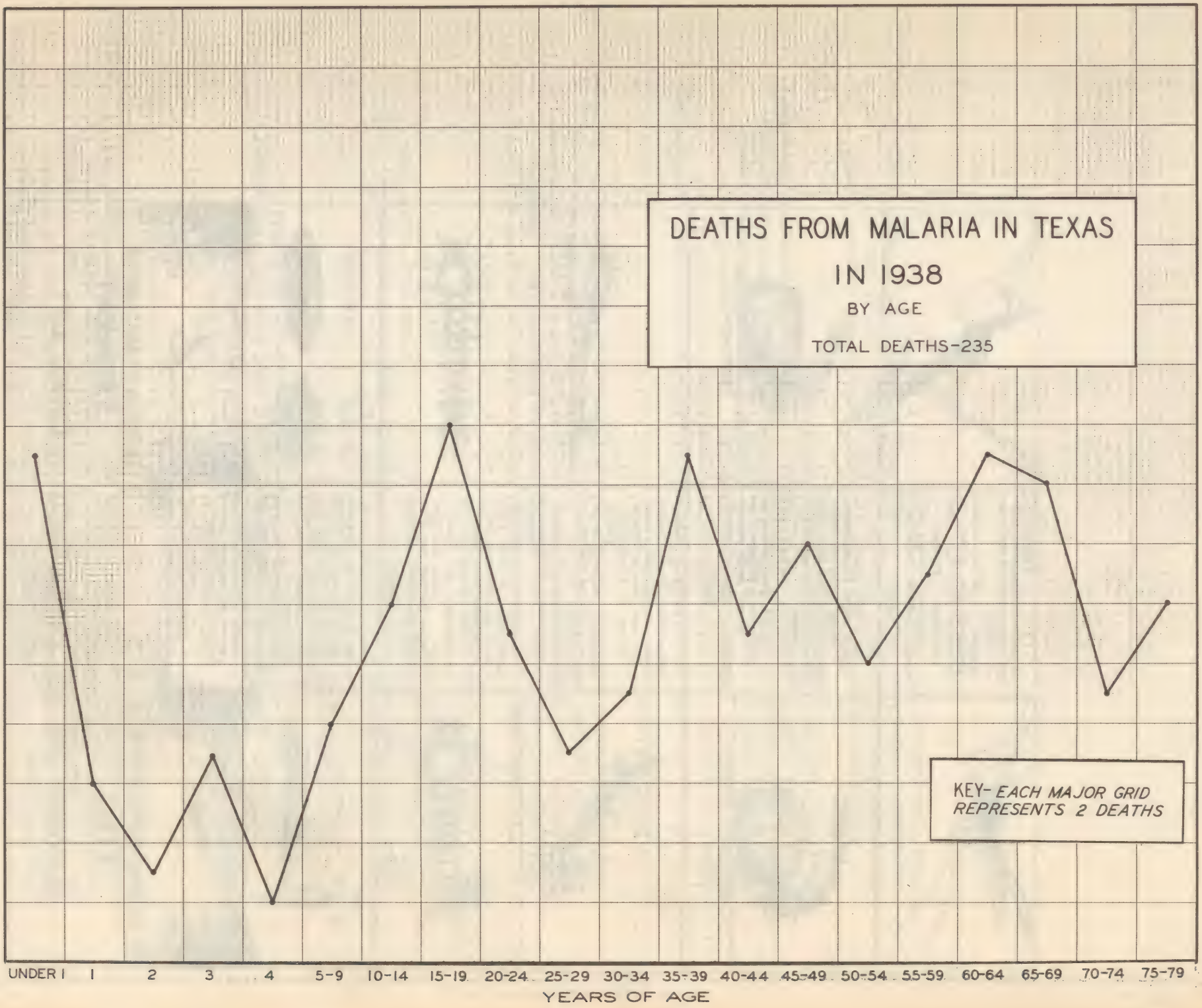
PUPAE

MOSQUITO PUPAE OR TUMBLERS DEVELOP FROM LARVAE.
←— ANOPHELES PUPAE HAVE WIDE BREATHING TUBES AND LONG HEADS.
CULEX —→
OR COMMON MOSQUITO PUPAE HAVE LONG NARROW BREATHING TUBES AND ROUND HEADS.
DURATION OF PUPAE STAGE: 1 TO 3 DAYS.



ADULT

THE ADULT MOSQUITO DEVELOPS FROM THE PUPAE.
←— ANOPHELES MOSQUITOES REST WITH THEIR BODIES AT AN ANGLE AND SOMETIMES PERPENDICULAR TO SURFACE.
CULEX —→
OR COMMON MOSQUITOES REST WITH THEIR BODIES PARALLEL TO SURFACE.


DEATHS FROM MALARIA IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS-235
KEY- EACH MAJOR GRID REPRESENTS 2 DEATHS
UNDER 1
1
2
3
4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
YEARS OF AGE

The history of malaria in Texas dates back to its earliest settlers. The vastness of malaria throughout the world, particularly in the tropical, semi tropical and temperate climates (of which Texas is in the latter two) makes it very difficult to incriminate any particular race of people or even the approximate century that the malarias may have been introduced in Texas. At this writing we do know that malaria is one among the five (5) leading communicable diseases in the Eastern and Southern sections of this state. The chart showing the deaths from malaria by age groups for the year 1938 indicates equally as much as any words can the wide range of fatal cases. While the chart shows the ages of only two-hundred nineteen (219) of the two hundred fifty-six (256) death certificates issued caused by malaria in Texas during 1938, the remaining thirty-seven were over eighty and under ninety-nine (80 to 99) years. This is very significant in that it shows deaths can and do occur from malaria in all age groups from birth to the age of ninety-nine (99) years. The mortality of malaria is estimated conservatively at one death to represent approximately each three hundred (300) active, acute cases of the disease. Since comparing the data available we find the year 1938 has the lowest death rate from malaria in Texas of any of the previous yearly records. We may assume from these data that approximately seventy thousand five hundred (70,500) cases of malaria occurred in Texas during 1938. These figures would indicate that approximately two per cent (2% of the inhabitants in the established malaria areas were afflicted with malaria during 1938. This is a very conservative estimate of the prevalence of malaria for the reason that many cases of malaria are very mild and are treated with home remedies and not seen by the family physicians, and therefore are not counted or even estimated in the tabulations on this chart. It is, however, the consensus of opinion among both the medical profession and laity alike that malaria is an impoverishing disease and it is an unnecessary disease. Malaria can be prevented; it can be controlled; it can be eradicated if enough concerted effort is exerted by a community.

The synonyms of malaria are (a) chills and fever; (b) swamp fever; (c) ague; (d) intermittent fever; (e) every other day or every third day fever. There are three separate malaria fevers as we know them today in the United States, and all three of the malaria fevers occur in Texas. They are Tertian malaria fever, Estivo-Autumnal malaria fever, and Quartan malaria fever. They each have their own individual causative organisms, and they do not mix or fight each other. It is not uncommon for a person to have two of these malaria fevers at one time, and in a few rare instances, all three malaria fever organisms have been found in a human in the same drop of blood collected for diagnostic examination.

The causative agents for all malaria fevers are the respective Tertian, Quartan, and Falciparum malaria plasmodia. Malaria plasmodia are animals that live, thrive, and reproduce in the red blood cells of man and in the bodies and salivary glands of female Anopheline mosquitoes. The only natural way man can become infected with malaria is through the bite of an infected Anopheline mosquito. And the only way a female Anopheline mosquito can become infected with malaria plasmodia is by biting a human that has adult malaria plasmodia in his circulating blood.

The parasitology of all three of the malarias is well defined and is a standard part of the laboratory work. The blood changes are the same in all three types of malaria, however, the toxicity differs in individuals as well as in each type of malaria:

(1) The penetration of red blood corpuscles by the animal parasites.
(2) Multiplication of the parasites and the concomitant destruction of the red cells in which they develop.
(3) Liberation of pigment into the blood plasma.
(4) The presence of free parasites and parasitized blood cells so altered as to act as foreign bodies in the circulation.
(5) A slight increase in white blood cells, chiefly phagocytes, as the macrophages, polynuclear and transitional forms of cells.
(6) The occurrence of melanemia, clumps and granules of melanin taken up by the white blood corpuscles, easily seen in stained specimens.

The entomology relative to all three types of malaria is the same, i.e., the same species of mosquito may carry any one of the three species of malaria plasmodia, while only the genus Anopheles (mosquitoes) have been found to be able to transmit malaria plasmodia to man. In the United States nine species of this genus (Anopheles) have been proven to be capable of successfully transmitting the disease either in the laboratory or in nature, or both. They are as follows:

(1)	A. quadrimaculatus	(4)	A. crucians	(7)	A. atropos
(2)	A. punctipennis	(5)	A. albimanus	(8)	A. barberi
(3)	A. pseudopuncitipennis	(6)	A. maculipennis	(9)	A. walkeri

For more detailed information about mosquitoes see article "Life and Habits of Mosquitoes", a regular Texas State Department of Health publication.

The epidemiology of malaria consists of the following:

a. Reported information of suspected cases of malaria and community investigations of factors involved.
b. Diagnosis completed only by laboratory confirmation.
c. To determine, if possible, whether or not the case is acute or relapse from a sub-acute stage or a chronic case of malaria.
d. Investigate and give study to possible sources of infection, such as contact with some other community within the last two or three weeks or with other cases of malaria in community.
Study the mosquito population in community where case or cases are present.
Study the housing and anti-mosquito measures in use by persons known to have been sick recently.
e. Complete laboratory examination of blood specimen of the suspected malaria patient.
f. The catching of an adequate supply of mosquitoes for identification and possible dissection purposes.
g. A compilation of data and a report of situation to proper authorities.

The diagnosis of malaria in cases where a laboratory is not available sometimes is quite difficult due to the fact that several diseases have similar physical signs in their incipiency. In a differential diagnosis of malaria by physical signs only, always consider the possibility of encountering one of the following diseases:

a. Dengue fever
b. Influenza
c. Heptatic abscess

d. Typhoid and possibly paratyphoid fever
e. Undulant fever
f. Tularemia
g. Early tuberculosis
h. Acute rheumatic fever
i. Septicemia

The pathology of malaria may be very simple and of short duration, or it may be serious and produce fatal results to the patient. The sequelae of malaria depends considerably more upon whether or not complications develop than upon the type of the malaria the patient is afflicted with.

The clinical portal of toxic or complicated malaria may be defined according to the pathological signs and physical exacerbations, as

1. Algid type
2. Pneumonic or congestive type
3. Bilious type
4. Comatose type
5. Choleraic and dysenteric types
6. Hemorrhagic or black water fever

Of greatest public health importance is, of course, the prevention of the spread of the disease. Second in importance is the assistance that can be given the community to prevent malaria carriers from infecting Anopheline mosquitoes, thereby endangering the community with the disease. It is highly important that all cases of malaria be treated by the family physician; that proper screening is done to prohibit mosquitoes from transmitting the disease; that mosquito eradication is conducted where practical and economical. When laboratory work is done modern malaria microscopy should be stressed, as well as thorough treatment to prevent the development of malaria carriers. When in doubt the City or County Health Officer should be consulted.

The first requisite in the prevention of malaria in any community is leadership by energetic, qualified public health officials - doctors, nurses, public health sanitary engineers and sanitarians.

The next step to be taken is the establishment of a local community plan which everybody may participate in. The plan should be broad enough to develop the malaria prevention problem along several lines, as persons possessing different qualities may unselfishly spend their talents for the common good of the community -- we find listed in the order of importance:

1. Proper advice by the family physician is the most profitable investment.

2. Employment of known measures for destroying larvae of anophelines and the eradication of breeding places of such mosquitoes.

3. Blood examination of persons living in infected centers to determine the incidence of infection. The individual with malaria parasites in his blood should be protected from bites of mosquitoes.

4. Screening sleeping and living quarters; use of mosquito nets.

5. Killing mosquitoes in living quarters.

6. Education of the public as to the mode of spread and methods of prevention of malaria.

TICK-BORNE DISEASES OF THE SOUTHWEST

The principal diseases transmitted to man by ticks in the Southwest are Relapsing Fever, Tularemia, and Rocky Mountain Spotted Fever.

Relapsing Fever

The period of incubation of Relapsing Fever is from two to twelve days, usually five to eight. There may be a prodromal stage of from a few hours to two or three days consisting of headache, loss of appetite, and lassitude.

The onset of this fever is acute with chills, aching of the head, back, and joints, nauseau and vomiting. The temperature rises from 98° F. to 103°-104° F. and is accompanied with an increase in pulse and respiration. The temperature remains high from five to six days, often with fluctuations of three to four degrees centigrade. During this period there is nausea and vomiting, dry and hot skin, light perspiration, and jaundice. Constipation may ensue, but diarrhea, nose bleeding, and hemorrhages from the intestinal tract, gums, and ears are most often observed. Delirium may be present but rarely stupor. Enlargement of the spleen and also albuminuria with rapidly developing anemia is common. Moderate granulocytic leucocytosis is present, and the spirochetes may be demonstrable among the granulocytes.

About the sixth or seventh day the patient apparently returns to normal. Within six or seven days after the afebrile state relapse again ensues. The symptoms of this second relapse are the same as those of the first, but they only last from three to four days, ending with crisis. A third and fourth relapse may occur; very rarely more than four relapses take place. Clinically it is difficult to differentiate between Relapsing Fever, Malaria, Dengue, Smallpox, Influenza, Yellow Fever, and the early stages of Plague. Weil's disease may also simulate this picture.

The following procedures are used in the laboratory diagnosis of Relapsing Fever:

(1) Demonstration of the presence of spirochetes in the blood during relapse affords a positive diagnosis of this fever. The spirochetes may be examined by direct wet smear under a cover glass, either by bright or dark field illumination, or a thick or thin smear may be stained with Giemsa's or Wright's stain.

(2) Blood from the infected individual may be injected intraperitonially in a white rat or mouse. Within four to six days spirochetes can be demonstrated in the blood of the mouse. They are not numerous in the peripheral blood, during the first few days of the disease.

The prevention of relapsing fever is very simple: The separation of man from the vector by the isolation of all convalescents and the protection of all persons from such vectors as lice and ticks.

Tularemia

Tularemia is an infectious disease produced by Bacterium Tularense. The etiological agent is present in infected rodents, especially rabbits. It is secondarily a disease of man, being transmitted from the animal reservoir to man by the bite of ticks, deer flies, or by handling the dead bodies of infected rodents. The incubation period of this disease is from one to ten days. The average period is three and one-half days.

The onset is very sudden and is characterized by chills, headache, vomiting, aching bodily pains, sweating, fever, and prostration. This disease manifests itself in four definite clinical types which are the Ulcero-Glandular, Oculo-Glandular, Glandular, and the Typhoid type. A pulmonary type might well be included.

Within forty-eight hours after the onset of the Ulcero-Glandular type, the patient complains of pains in the area of the lymph glands which drain the site of infection. These glandular pains are accompanied by swelling of the glands involved, which are only those of the immediate vicinity of the site of infection. These glandular pains are preceded by a papule at the site of the insect bite, which breaks down and leaves a ragged ulcer. This primary site is often found on the back of a finger.

The skin area over the infected glands is reddened. The glands often break down and discharge purulent material. Incision of fluctuant glands is usually ill advised, as it results in chronic sinuses which prolong the course of the illness.

In about half the cases these glands do not break down, but remain hard, palpable, and rather tender for two or three months, gradually returning to normal. The lymph glands other than regional are usually only slightly involved.

Subcutaneous nodules, varying in size from that of a pea to one centimeter in diameter and numbering from two to thirty are sometimes present on the anterior aspect of the forearm and hand. The nodules are found along the blood vessels from the ulcer on the finger to the axillary glands.

Loss of weight, extreme weakness, recurring chills, sweating, and prostration are characteristic of the active stage which lasts from two to three weeks.

In general, the Ocula-Glandular type presents the same symptoms as the above mentioned type, except that the primary lesion is in the conjunctival sack instead of the skin.

The papules usually occur on the lower lid. The eye manifests irritation, redness, and lacrimation. The preauricular, parotid, submaxilliary, anterior cervical, and, in severe cases, the axilliary glands, are infected.

The glandular type is characterized by the absence of a primary lesion at the site of infection, also by the marked swelling of the regional lymph nodes. This type resembles the Ulcero-Glandular.

The Typhoid type of Tularemia is chiefly characterized by the absence of a primary lesion and the swelling of the lymph nodes. The general symptoms of this type resemble those of Typhoid Fever. The onset and duration of this type is the same as that of the Oculo-Glandular type.

Fever is always present in all four types of Tularemia. Patients show constantly an initial rise in temperature, remission, and secondary rise. Following the initial rise there is a remission of one, two, or three days which is followed by a rise to the original height. After this there is a gradual decline to normal which may take two or three weeks to gain.

Convalescence of Tularemia is very slow, and patients in this stage are often unable to work after six months or even a year. This weakness is very characteristic of Tularemia.

Relapses of fever lasting six to eight days have occurred as late as ten months after the patient had apparently recovered.

The clinical diagnosis of Tularemia is easily made, providing the physician will keep in mind the following points:

(1) A history of the patient having dressed a wild rabbit or being bitten by a tick.

(2) A primary lesion of the skin in the form of a papule developing into an ulcer.

(3) Persistent glandular enlargement in the regions draining the primary lesion.

(4) A fever of from two to three weeks' duration and of an undulating nature. These things should be kept in mind to prevent the clinician from calling it "flu", septic infection, typhoid fever, or pneumonia; the serologists from calling it undulant fever on account of the cross-agglutination with melitensis and abortus, or typhus because of a high titre in the Weil-Felix reaction; the pathologist from calling it tuberculosis because of the lesions produced in the lymph glands, and the dermatologists from calling it sportrichosis due to the subcutaneous nodules.

The bacteriological diagnosis of Tularemia is made to verify the clinical diagnosis after the physician has recognized one of the above four points in his patient. The procedure may be one or both of the following methods:

(1) By obtaining an agglutination of Bacterium Tularense with blood serum taken from the patient not earlier than the second week of illness and noting an increase in the agglutination titre of the serum taken a few days later or on the third week of the disease.

(2) By isolation of Pasteurella tularensis from guinea pigs inoculated with material from the primary lesion, or from the enlarged glands or the blood of the patient. Microscopic examination of the cover-glass preparations taken direct from the patient is useless. After subcutaneous inoculation of the guinea pig, death occurs in five to eight days; at post-mortem there is a whitish membrane-like area at the site of inoculation. There is a bubo, generally in the cervical axilliary, or inguinal region, containing dry, yellowish caseous material; the spleen is greatly enlarged, very dark in color and contains yellowish white discrete, caseous granules up to one M.M. in diameter, projecting slightly above the surface; there are numerous granules in the liver; the lungs are rarely involved. The organisms are present in enormous numbers in the spleen, in smaller numbers in the liver, bubo, and heart's blood.

The prevention of Tularemia is carried out in the following ways:

(1) Cooking thoroughly any wild animal used for food, especially rabbits.

(2) The use of rubber gloves when working in laboratories where necropsy on infected animals is being performed.

(3) The use of rubber gloves by cocks, hunters, and market men in dressing rabbits.

(4) Use of an immune person for dressing rabbits where possible.

(5) Freezing the rabbits for thirty days or longer.

(6) The use of the ordinary disinfectants.

The only treatment for Tularemia is symptomatic. Complete rest in bed is probably the best single measure. Excision or even incision of the glands is not advised. No drug, preventive vaccine, or serum has been found yet.

Rocky Mountain Spotted Fever

Rocky Mountain Spotted Fever is an acute, endemic, febrile disease which is transmitted accidentally to human beings by the bite of an infective wood tick, Dermacentor andersoni.

The prodromal and early symptoms of Rocky Mountain Spotted Fever resembles closely those of the other acute, eruptive, febrile diseases and, as in those, vary in degree; therefore, it is difficult to establish an early diagnosis in the disease. During the progress of this disease the following symptoms appear: a short prodromal period lasting two or three days during which the patient has a general malaise and chilly sensation. This is frequently followed by distinct chills, pronounced headache, and muscular pains. Rash and rose colored eruption are seen early. This rash and rose colored eruption occurs on the face, neck, and upper thorax. From twenty-four to forty-eight hours later in the wrist, forehand, ankle, and lumbar region of the back appears the characteristic petechial eruption, which is rose-red in color, changing rapidly to dark bluish-red. This is the result of hemorrhages from small blood vessels and will not disappear on pressure. The petechial increase in size, become purpuric and are accompanied by sloughs on the dependent parts of the body in the more severe cases. Not infrequently sloughing of the soft palate and scrotum appears in the human. The tongue has a yellowish white coat and the conjunctiva shows a yellow tinge in the later stages of the disease. Delirium is rare, the mind usually being clear. Extreme lassitude is the rule, constipation and albuminuria are common. The temperature is high from seven to ten days in uncomplicated cases, while in complicated cases it may be prolonged. The respiration and pulse are rapid, with the latter out of proportion to the degree of temperature, due to the toxemia. Convalescence is slow but complete, with the temperature dropping rapidly.

The clinical diagnosis of Rocky Mountain Spotted Fever is made from the history and surroundings of the case and the character of the eruptions. It is usually easily arrived at after the petechial eruption has appeared, except in rare cases where the history doesn't exclude possibility of typhus fever infection. In such cases, blood inoculation into guinea pigs will easily establish the diagnosis because these two diseases are distinguishable in this animal both by character of lesions and by absence of cross immunization. The history of the tick bite is usually found to be from three to twelve days previous to the onset of the

sickness. When there is no history of a tick bite and the tick has dropped off, the site of the bite is hard to locate. This is the habit of the tick Dermacentor andersoni. In some cases slightly elevated rose colored and macular eruptions appear on the face, neck, hands, and upper thorax before the characteristic petechial eruption is visible. These cases may easily be mistaken for measles or early smallpox or other exanthematous febrile diseases.

If a careful examination is made, the primary eruptions of Rocky Mountain Spotted Fever will not be confused with early appearing erythemia or the diffused red-blush of scarlet fever, or with the rapidly changing papules and vesicles of chicken pox, or with the localized abdominal rose spots of typhoid fever. From the standpoint of subjective symptomatology, especially as regards headache and myalgia, only typhus and smallpox are likely to be comfused with Rocky Mountain Spotted Fever.

In the biological diagnosis of Rocky Mountain Spotted Fever, a blood inoculation from the infected individual is made intraperitonially into a male guinea pig. This brings about swelling of the scrotum and inguinal region of the animal and hemorrhages of the brain which produce death in the animal, in the case of Rocky Mountain Spotted Fever. In the case of Typhus, swelling of the scrotum and inguinal region takes place but the animal never dies, thus differentiating between the two diseases. Agglutination tests using non-specific antigen, proteus OX-19 and OX-K, is also used in diagnosing this disease. The same diagnostic test is used in typhus fever. An early high titre with immediate and rapid increase is diagnostic of typhus and differentiates it from Rocky Mountain Spotted Fever, which infrequently has this early high titre and rapid increase.

There are three measures which we, as individuals, may use to prevent spotted fever:

(1) Avoid ticks.

(2) Remove ticks from the person as early as possible.

(3) Be vaccinated.

On camping trips, if it is necessary to sleep in the open, care should be used in selecting a site for placing the bed, as ticks will crawl into a bed laid on the ground. Since ticks are usually most numerous where rodents are most abundant, areas well populated with rodents should be avoided. The safest camping ground is undoubtedly in standing timber where low vegetation is scanty. Proximity to trails and old roads should be avoided. In sage-brush sections, avoid the sage-brush. Avoid brushy areas along streams as camping grounds. The dog tick is far more likely to be present along the course of streams than is the wood tick. Persons should be especially watchful when walking along trails. Ticks tend to concentrate on vegetation along the sides of trails and in the bushes along the edge of wooded areas. Similarly, vegetation along roadsides and grassy strips in the middle of little used roads are often very dangerous. It is especially desirable to watch the clothing when following trails or old roads.

In the prevention of tick bite, the first precaution is the wearing of such clothing as will prevent ticks from getting underneath. This may be accomplished to a considerable extent by wearing high boots, leggings, puttees, or socks that are worn outside the trousers legs.

With such precautions taken, most ticks will crawl up the outside of the clothing and can be removed from the neck when contact with the skin makes their presence known. Passing the hand over the neck occasionally to feel for ticks is a good habit to acquire.

Ticks are far more likely to secure a hold on rough clothing than on clothing of smoother texture. There are advantages in both, however. Fewer ticks secure a hold on smooth clothing; but, on the other hand, on cloth with a heavy nap their movements are impeded and are necessarily much slower. If the legs of the trousers are carefully watched, most ticks can be picked off soon after they catch hold.

In spite of precautions, however, a certain number of ticks will reach the body through the various openings in the clothing. It is therefore important that the above precautions be supplemented by the examination of the inside of the clothing and of the body. Since ticks seldom attach immediately (unless late in the season), and are seldom infectious until after having been attached for a few hours, such examinations made twice each day (early afternoon and on retiring) should ordinarily be sufficient. In heavily tick-infested areas, however, or in sections known to be particularly dangerous, more frequent examinations should be made. When retiring, a complete removal of clothing is desirable. Both clothing and body should be examined carefully and, if possible, any clothing not worn at night should be so placed that any undiscovered ticks will be unlikely to crawl from the clothing to the bed. If two or more persons are together, they should assist one another in the examination. If the person is alone, the back and other portions of the body that cannot be seen should be explored with the hands, paying particular attention to the hairy portions.

Ticks may be removed from man and the domestic animals with the fingers, but a better plan is to use a pair of small forceps or tweezers. With these the tick may be seized by the head, close to the skin, and easily removed. There is no danger of leaving the tick's head imbedded in the skin. Care should be exercised against crushing the tick, as the contents of infected ticks are dangerous. After removing or handling ticks, the hands should be washed thoroughly with soap and water.

Two or three inoculations of the tick emulsion vaccine give a degree of protection usually sufficient to last through one tick season, but the immunity apparently is not permanent. Occasional cases of spotted fever have developed in vaccinated persons, but the vaccine apparently lessens the severity of the disease and seems to insure recovery. For its full protective value the vaccine should be taken at least 10 days before exposure to tick bite.

THE RICKETTSIAL DISEASES

Introduction

The Rickettsia diseases are acute infections characterized by high fever, a maculo-papular eruption which becomes petechial, headache, and stupor. They are all transmitted in nature by arthropods (lice, ticks, fleas, or mites) either directly from man to man or from rodent reservoirs to man. These diseases may be divided in two general groups, typhus fever and Rocky Mountain Spotted Fever. They appear to be variants of the same disease.

Causative Organisms

The Rickettsiae are very minute, pleomorphic, cocco-bacillary organisms, smaller than the ordinary bacteria. They stain well only with the Romanowsky stains (Giemsa, Wright, etc.). They have never been cultivated on artificial media, but have been cultivated in tissue cultures, and on chick embryos. They are too large to pass through a Berkfeld filter. In man they multiply mainly or only in endothelial or mesothelial cells, in the arthropod mainly or only in the epithelial cells of the intestine.

The Rickettsiae were discovered in 1910 by Ricketts and Wilder in the intestines of lice which had fed on typhus patients in Mexico. Later Ricketts found similar bodies in the blood of patients with Rocky Mountain Spotted Fever, and Wolbach found them in endothelial lesions of experimental animals.

The question frequently arises as to whether the Rickettsiae are bacteria or "virus particles". The question cannot be answered at present but the following seems to be a fairly tenable view.

"The Rickettsiae are living organisms whose obligate intracellular parasitism indicates a primary inability to metabolize foodstuffs"

TYPHUS FEVER

Causative Organism. - Rickettsia prowazeki.

Synonyms. - Camp Fever, Jail Fever, Ship Fever, Spotted Fever, Famine Fever, Typhus Exanthematicus, Tabardillo (Mexico), Brill's Disease (New York).

Varieties.

1. Epidemic Typhus, more severe, higher mortality, transmitted by the louse (Pediculus humanus); occurs in epidemics usually starting in winter; associated with poverty, filth, war and famine; rare in the tropics.

2. Endemic Typhus, less severe, very low mortality, transmission by rat flea, affects higher class of people or people working with grain or cattle or in clearing underbrush; not epidemic, but occurs most often in warmer months, common in the tropics. Endemic typhus is probably a variant of epidemic typhus modified by passage through the rat. This is the type of typhus fever in Texas.

EPIDEMIC TYPHUS

Historical

The disease probably existed in ancient times but was first carefully described during the sixteenth century by two Italian physicians. From that time on it is known that typhus caused frequent devastating epidemics in' Europe and was one of the chief causes of death until the middle of the nineteenth century. It was first clearly differentiated from typhoid fever in 1837. In the United States it has never reached serious epidemic proportions, though introduced often by immigrants. During and after the World War there were terrible epidemics in the Balkan States, Poland and Russia, but there were very few cases in the armies of Britain, Germany, France or the United States. In 1909 it was proved that the disease is transmitted by the body louse.

Geographical, Seasonal Incidence and Transmission

These three factors are related because of the biology of the transmitting agent, the body louse (Pediculus humanus, var. corporis). It lives close to the skin in the clothes, and favors a temperature of about 90°F. It stays closer to the skin in cold weather, but crawls outside in warm weather, or if its host has a high fever as in typhus. It is less abundant in summer when thin or no clothing is worn. Having become infected from a typhus patient in the period of onset, it leaves him in the high fever period and seeks a cooler, normal host. The louse feeds only on man. Epidemics, therefore, occur usually in temperate or cold climates, start in winter, and die out in summer. The epidemic disease is now found only in backward countries where pediculosus vestimente is common. The louse, once infected, remains so throughout its life, which lasts up to 45 days. The virus has been recognized in the louse only in the intestine. It has not been determined whether infection is acquired direcly from the bite of the louse, or only from scratching after a bite, louse excreta having been deposited on the skin. Many medical attendants become infected during epidemics, often without knowledge of having been bitten by a louse. Either the louse is very elusive, or infection may be acquired from louse excreta transferred from the body or clothes of a patient.

Mortality

Varies greatly (2-50 per cent) in different epidemics and different population groups. Higher social classes when infected usually have a higher mortality.

Prevention

Depends entirely upon anti-louse measures.

1. Delousing

The louse and its eggs are easily killed by a dry heat of 55°C. for five minutes, or by a moist heat of 70°C. for 30 minutes. Of chemicals, kerosene is much the most efficient. Cyanide, naphthalene, carbon disulphide and ammonia are also used. The louse is the most resistant of insects to fumigation. Ten ounces of sodium cyanide per 1000 cubic feet of air space with two hours exposure is required. Sulphur dioxide and formaldehyde gases are of no value. For clothing, heat is usually used. The "Serbian Barrel" was improvised to provide live steam. Closed shacks with a wood fire burning inside are also used. Pressing seams of clothes with a hot iron may be employed, as eggs are usually laid on loose ends of cotton or wollen threads. Eggs are

sometimes attached to body hairs (the head louse P. humanus var capitis, may also transmit the disease). A typhus patient or healthy host of the louse is best deloused by

(1) shaving all hair

(2) a bath of soap and kerosene

(3) an entire change of clothing

(4) female patients with head lice should have the hair washed first with Tincture of Delphinium and later with hot vinegar to loosen the eggs, which should then be combed out with a fine-toothed comb.

After thorough delousing and bathing, a patient is not a source of danger to others, and can be treated in a general ward.

2. Protection Against Lice

Attendants handling typhus patients or suspects must wear a louse-proof garment. This should be open only at neck and wrists and should be provided with feet and soles so as to cover the shoes. Wrists should be tied with tapes or better fastened by adhesive plaster. Cotton or rubber gloves tucked into sleeves of gown should be worn. Garment should be tied snugly about neck. such precautions, though extreme, have been found necessary during epidemics.

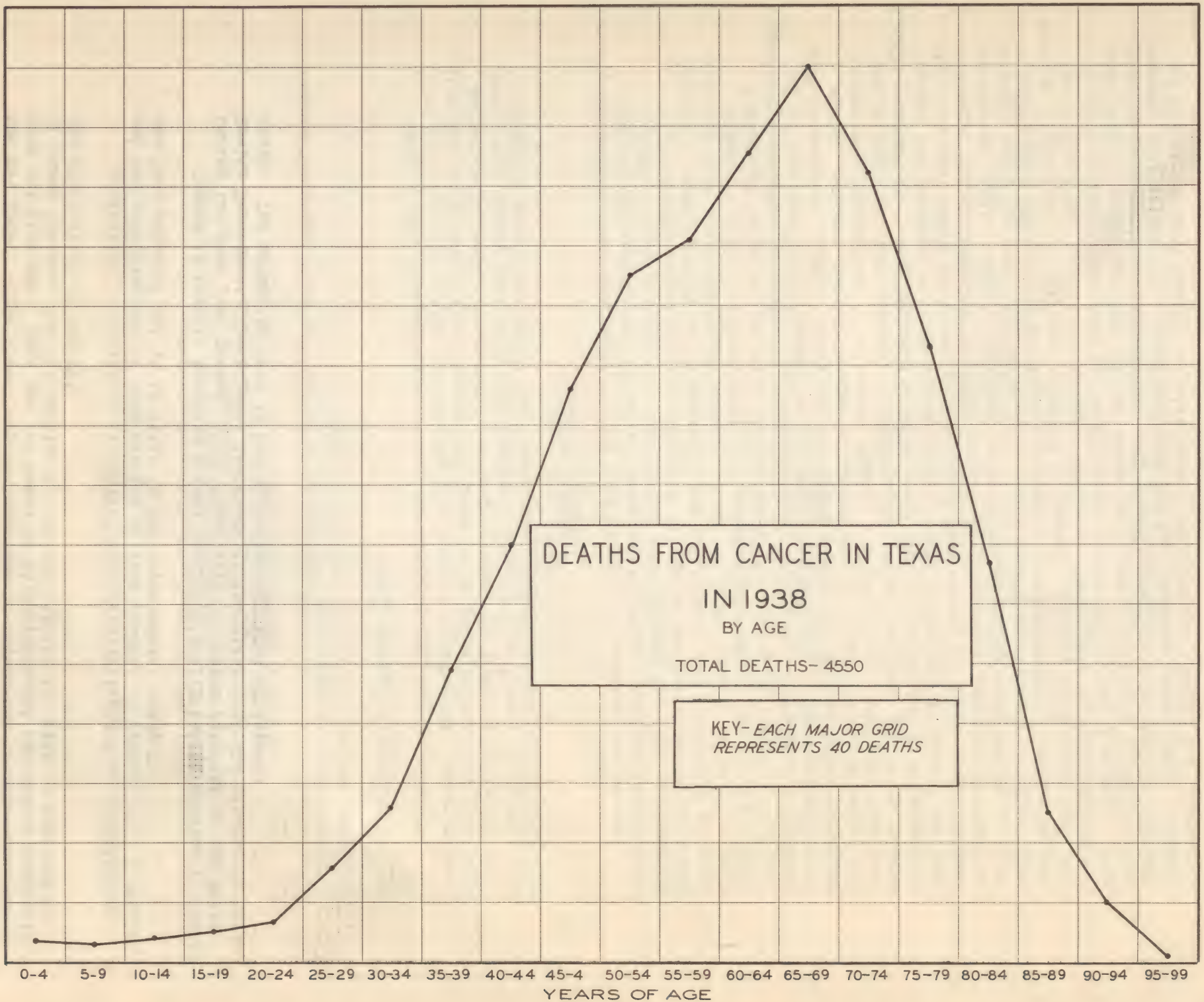
ENDEMIC TYPHUS

The Mexican Indians are said to have had a typhus-like fever before the discovery of America. After the conquest by Cortez the disease was called "Tabardillo," the old Spanish name for typhus. It has continued in Mexico until the present time and exists in Peru, Bolivia, Chile and Argentina. Ricketts studied it and first described "Rickettsia" in Mexican cases. In 1923 cases similar to it were found in the Rio Grande Valley and since then in practically all of the Southeastern States; the incidence has slowly increased. They have occurred in people not infested with lice, in the upper and middle classes, and especially in people handling grain. A number of cases have occurred in professional men. The disease occurs in the tropics at all seasons, in the temperate zones only in the warmer months of the year. Direct transmission from man to man does not occur. As the result of epidemiological studies, it was concluded that there was probably a rodent reservoir of the disease, presumably rats or mice, and that man became "accidentally" infected from the bite of some ectoparasite of the rodent, either flea, mite or possibly tick. In February 1931, Dyer and Badger of the United States Public Health Service reported the production of typhus fever in guinea-pigs injected with ground-up fleas from wild rats. In July 1931, Mooser, Castenada and Zinsser reported the production of typhus fever in guinea-pigs by the injection of an emulsion of brain tissue from black rats caught in a prison in Mexico City. In October 1931 Dyer and other reported the experimental transmission of typhus in laboratory rats by the bite of the rat flea, Xenopsylla cheopis. Meanwhile Shelmire and Dove, in June 1931, reported the transmission of the disease in guinea-pigs by the bite of the tropical rat mite, Liponysus bacoti. They also showed that the causative organism could be transmitted to the next generation of mites through the egg. They believe that the epidemiology of the disease favors mite transmission rather than flea transmission. Their work, however, has not been confirmed. In October 1931, Mooser, Castenada, and Zinsser reported the transmission of typhus from rat to rat by

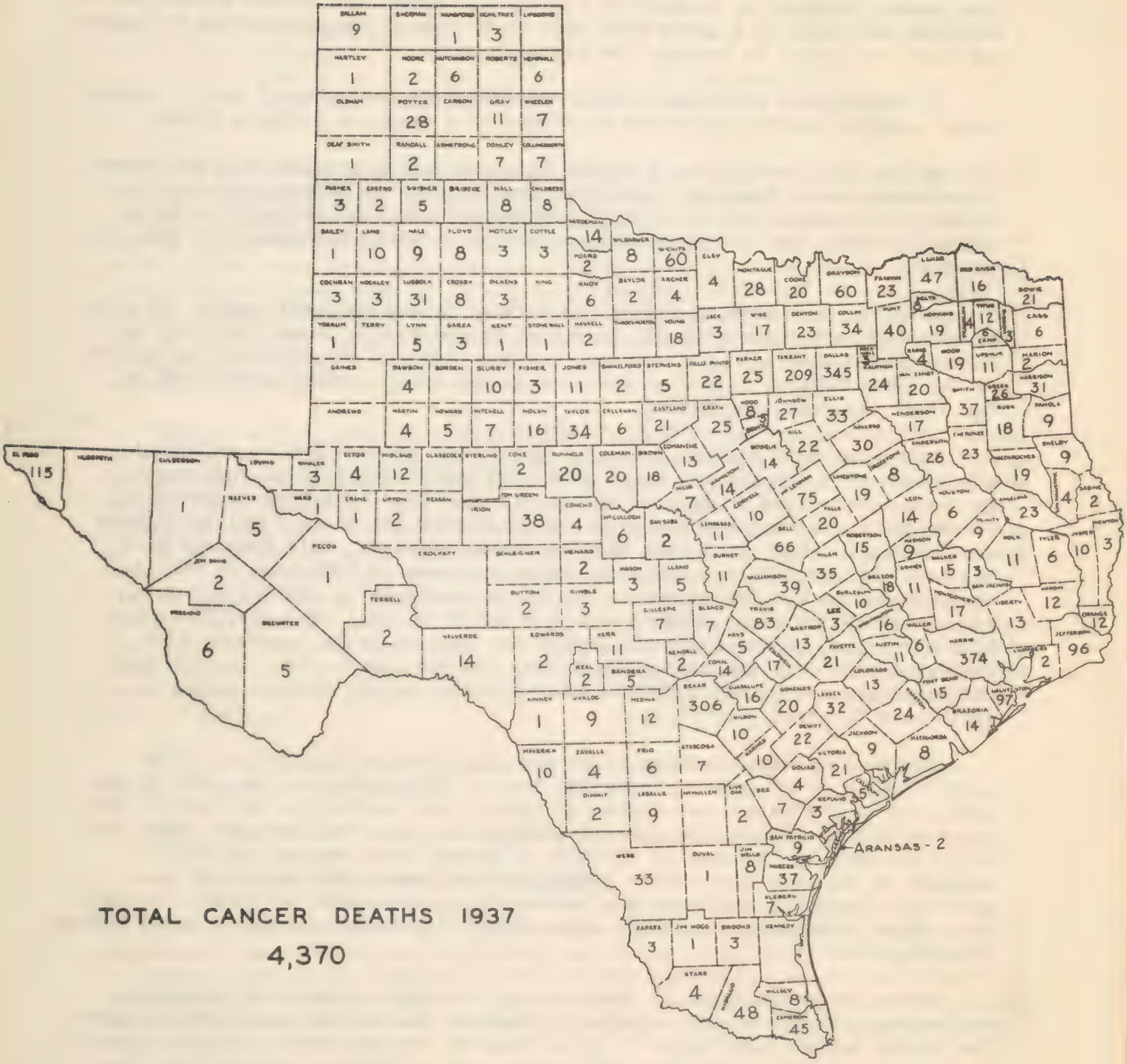
the rat louse. This insect may be important in rat-to-rat transmission, but it does not bite man. Thus the rat seems to be incriminated again as a reservoir of human disease, and the eradication of the disease can best be achieved by the eradication of the rat.

Prevention. - Avoidance of contact with rats, rat-proofing of buildings and the extermination of rats are the obvious methods of prevention. A vaccine has been prepared but it is still in the experimental stage.

Typhus is rapidly becoming a very important public health problem in Texas. During the year just passed, 1939, there were 538 cases reported to the State Health Department, the highest incidence in the history of the disease in this state. The mortality rate is now approximately that of typhoid.


DEATHS FROM CANCER IN TEXAS
IN 1938
BY AGE
TOTAL DEATHS- 4550
KEY- EACH MAJOR GRID REPRESENTS 40 DEATHS
0-4
5-9
10-14
15-19
20-24
25-29
30-34
35-39
40-44
45-4
50-54
55-59
60-64
65-69
70-74
75-79
80-84
85-89
90-94
95-99
YEARS OF AGE

CANCER MORTALITY
IN TEXAS
1937



TOTAL CANCER DEATHS 1937

4,370

How shall we protect ourselves against cancer, the greatest of all the natural hazards in the adventure of living? Generally, like old age, cancer' is the result of the wear and tear of the tissues, due to the natural stresses of living. Unlike senility, cancer attacks the young much more frequently than is commonly realized and it plays the greater havoc the younger the subject. If an ounce of prevention is generally worth a pound of cure, who may measure the value of cancer prevention? Yet prevention does not form a prominent chapter in the literature of cancer. It has not been dignified, like cancer therapy, by decades of acrimonious debate in medical societies. Nevertheless there is a great deal more to be done in the prevention of cancer, and most of it must be done by the person himself.

For many years the cancer death rate has been rising until now it stands second only to heart disease and accidents as a cause of death in Texas.

We are all more or less familiar with the cancer problem from the scientific standpoint. That is, it is produced in areas or organs subjected to chronic irritation, that it is more predominant in later adult life, it is extremely fatal, and the treatment is usually surgical combined with some ray therapy.

Also, we have much of the cancer problem from a national angle. We wish to discuss the problem with you Texans as it relates to Texas. We want to review the records with you and tell you the story of the influence of cancer on the death rate of Texans as revealed by the death certificates filed in the State Department of Health.

Let us go back sixteen years to 1923. That year we had an estimated population of 5,095,516 and there were 1605 deaths reported as from cancer. To avoid a long list of figures we may say that there has been a steady increase in cancer deaths until 1938, the last year for which complete figures are available, when there were 4550 reported. However, this does not give the true picture. There were more people in Texas in 1938 than in 1923 to be subjected to the cancer menace, so it is necessary to use another yardstick. In 1923 sixteen hundred and five out of a total of 5,059,632 people died of cancer, or for every 100,000 of our population 31.7 persons died. This we speak of as the cancer death rate for that year. Now, in 1938 our estimated population was 6,259,000 and 4550 died establishing a cancer death rate for that year of 72.6.

There are several reasons as to why cancer has an increasingly high death rate, one, of course, being the lack of information on the part of the public as to the nature and course of cancer, and another is the fear of the disease and the tendency to put off medical care for fear that the physician will find that the disease or trouble is in reality a cancer, and still another is the fact that public health efforts toward the control of communicable deaths in childhood has prevented a great number of deaths at this age. Thus, furnishing a greater population in those age groups in which cancer often appears.

Going back over these 15 years month by month, we see no evidence of any seasonal variation and reports by counties show no sectional predominance. The deaths appear distributed in proportion to the population except in centers where hospitals drew from wide areas, and efforts at distributing these deaths back to the community of origin further bears out the conclusion that

no areas are more affected than others. Although all elements of our population are affected by cancer it appears that the white race is slightly more susceptible than are the other races. They who comprise 73.5 per cent of the population accounted for 83.5 per cent of cancer deaths and all other races 16.5 per cent. The negro race which comprises 14.7 per cent of the population has 11.5 per cent of cancer, and the Mexicans which comprise 11.7 per cent only account for 5 per cent of cancer.

Let us look at the problem from the standpoint of the organs of the body most usually affected. We find that cancer attacks any tissue or organ of the body, but fatal cases occur in order of their frequency in the following manner:

1. Intestinal tract
2. Stomach
3. Uterus
4. Liver
5. Male G-U organs
6. Breast
7. Skin
8. Mouth cavity
9. Respiratory system
10. Female Genital organs

We find that cancer deaths appear in the first year of life and gradually increase in number up to age 25 where it plays a dominant part in the total causes of deaths through the remainder of life's span, reaching its peak during the age period of 65 to 69 and rapidly declining to the age period of 95 to 99. This study brings out one fact which is little considered and that is that after the first year of life cancer remains one of the fifteen leading causes of death throughout our existence. Conclusively illustrating the fact that no age is free from the menace and that any program to be wholly successful must include the total population instead of just those age groups where cancer deaths appear in great numbers.

Cancer as a disease is a public health problem and responsibility because it affects relatively large numbers of people, because its frequency as a cause of death is on the increase and because systematized social action seems necessary in solution of the problem. An analysis of the foregoing statement certainly reveals that any State Department of Health should be vitally concerned in the promotion of a cancer control program, since the work of such an organization is concerned with the prevention and control of disease and the promotion of positive good health among citizens of the state. There is some doubt as to just how far the State Department of Health of Texas should go into the promotion of such a program. We are, however, convinced that the following three essentials of cancer control should be carried on in every community in Texas and should be to a certain degree supervised through the State Department of Health:

1. A dissemination of information to all the public concerning the early symptoms of the disease.

2. Provisions for early diagnosis by competent specialists.

3. Adequate facilities for treatment.

The function of the State Health Department is to disseminate freely throughout its jurisdiction a thorough knowledge as to early warning signs of cancer. Educational material, if properly submitted, may be supplied without the danger of developing a cancer phobia. Such knowledge should be common among all people just as the knowledge concerning the value of anti-toxin for diphtheria.

It is an evident fact that something needs to be done in Texas if we

expect to make any progress in reducing the death rate from this dreaded disease. Since the Texas State Department of Health is now operating on a very limited appropriation, it is not difficult to understand why we have been confined to only a limited educational phase of a cancer control program.

In carrying forward this educational program, the State Department of Health has utilized newspapers, radio stations and free literature through which certain salient points concerning the disease are impressed upon the general public. The ordinary danger signals such as lumps in the breast, unexplained indigestion, loss of weight, chronic ulcers, pigmented moles, etc., are called to the attention of those who were interested and due emphasis has been placed upon the wisdom of periodic and thorough medical examination of individuals in whom these danger signals have appeared. The Department has also repeatedly called to the attention of the general public that hope for recovery from cancer depends upon early diagnosis and appropriate treatment.

Among the difficulties confronting our educational program are lack of interest on the part of the public and human dread of learning that cancer is present. In many instances, none of the danger signals show themselves until the condition is far advanced. Many physicians are inadequately equipped by knowledge, experience, and facilities to make a complete examination and diagnosis, or, in certain types of cancer, do not have necessary equipment for adequate treatment of the case. Finally, it must be admitted that even in the best circumstances there are definite limitations as to what can be done for the patient.

The degree to which the State Department of Health will participate in the future program of cancer eradication will depend largely upon finances, public interest, and public demand. We desire to impress upon you that we stand ready to assist the American Society for the Control of Cancer and every practicing physician in any possible way in their united effort to control this great killer of our people. At all times the work of the Department is planned to be in close harmony and accord with the organized members of the medical profession. The question of Cancer Control must be approached in much the same manner as we have approached those of typhoid fever, diphtheria, malaria and as we are now approaching the question of syphilis control. We have now instituted a program whereby cancer diagnostic service is available to physicians of Texas through biopsy studies at the State Laboratory.